

CIVIL ACTION NUMBER 5:96CV91

IN THE UNITED STATES DISTRICT COURT  
FOR THE EASTERN DISTRICT OF TEXAS  
TEXARKANA DIVISION

THE STATE OF TEXAS  
VS

THE AMERICAN TOBACCO COMPANY; R.J. REYNOLDS  
TOBACCO COMPANY; BROWN & WILLIAMSON  
TOBACCO CORPORATION; B.A.T. INDUSTRIES,  
P.L.C.; PHILIP MORRIS, INC.; LIGGETT GROUP,  
INC.; LORILLARD TOBACCO COMPANY, INC.; UNITED  
STATES TOBACCO COMPANY; HILL & KNOWLTON, INC.;  
THE COUNCIL FOR TOBACCO RESEARCH - USA, INC.  
(Successor to Tobacco Institute Research  
Comittee); and THE TOBACCO INSTITUTE, INC.

VIDEOTAPED

ORAL DEPOSITION

OF

WILLIAM SAM SIMMONS

August 15, 1997

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## ANSWERS AND DEPOSITION OF WILLIAM

SAM SIMMONS, produced as a witness at the instance of the Plaintiff, taken in the above-styled and numbered cause on the 15th day of August, 1997, at 9:00 o'clock a.m. before Amy Doman, a Certified Shorthand Reporter in and for the State of Texas, at the offices of Womble, Carlyle, Sandridge & Rice, located at 200 West Second Street, in the City of Winston-Salem, County of Forsyth, State of North Carolina in accordance with the agreements hereinafter set forth.

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## A P P E A R A N C E S

MR. JEFFREY S. THOMPSON  
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Houston, Texas 77017-5001

APPEARING FOR THE PLAINTIFF

MS. MARILYN R. FORBES  
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Washington, D.C. 20005-2088

APPEARING FOR THE DEFENDANT

Also Present: Mr. David George, Videographer

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**P R O C E E D I N G S**

MR. THOMPSON: I think we're taking these according to the Eastern District Rules, which means reserving all objections except as to privilege until time of trial. I guess that's it. Making it go a lot easier.

THE VIDEOGRAPHER: We're on the video record.

**WILLIAM SAM SIMMONS,**  
the witness hereinbefore named, being of lawful age and being first duly cautioned and sworn in the above cause, testified on his oath as follows:

**EXAMINATION**

**BY MR. THOMPSON:**

Q. Sir, could you please state your name for the record?

A. My name is William Samuel Simmons.

Q. And Mr. Simmons, are you currently employed?

A. I am.

Q. By whom?

A. The R.J. Reynolds Tobacco Company.

Q. Okay. And how long have you been at R.J. Reynolds?

00:16 1 A. I joined the company in December  
2 of 1984.

3 Q. So what's that, 13 years?

4 A. Pushing 13 years.

09:16 5 Q. Okay. What's your position  
6 currently?

7 A. I am a principal scientist in the  
8 research and development department.

9 Q. And as principal scientist, what  
09:16 10 are your duties?

11 A. I have had responsibility for a  
12 number of years for the -- the science around  
13 smoking and health issues.

14 Q. And when you say you've had  
09:16 15 responsibility for smoking and health issues,  
16 what does that mean?

17 A. In December of 1986 -- excuse me,  
18 excuse me. April of 1986 I became -- I had  
19 the title director of smoking and health. And  
09:17 20 as such, it was my job to review the  
21 scientific literature with respect to smoking  
22 and health issues.

23 Q. Okay. And other than reviewing  
24 the scientific literature, what -- what did  
09:17 25 you do in that capacity as director of smoking

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09:17 1 and health?

2 A. One of the responsibilities was to  
3 stay up-to-date with respect to the latest  
4 scientific studies published in the peer  
09:17 5 review literature and keep my management  
6 advised as to new studies, their relevance to  
7 the industry, their significance, and so  
8 forth.

9 Q. Were you empowered to make any  
09:18 10 recommendations in that regard?

11 A. The -- I'm not -- I'm not sure  
12 what you mean. I recommended and executed  
13 certain types of studies, scientific studies  
14 for which I had a budget to contract outside  
09:18 15 work.

16 Q. Okay. In your capacity as  
17 director of smoking and health, was one of  
18 your responsibilities to be a spokesman for  
19 your company on those issues?

09:18 20 A. I don't know that that was written  
21 down. It may have been in my job description,  
22 but it's been a long time, and I don't know  
23 that that was actually written in. I  
24 understood that as the most knowledgeable  
09:19 25 company -- person in the company on certain

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00:19 1 topics, I might be required to give testimony,  
2 if that's -- I'm not sure what you're  
3 referring to.

09:19 4 Q. Well, at least in your capacity as  
5 director of smoking and health, I guess what  
6 I'm asking is, did you understand your job to  
7 be that you would be asked on occasion to make  
8 public statements regarding the company's  
9 position on smoking and health issues?

09:19 10 A. I don't recall that as being in  
11 the job description. What I do recall is  
12 that -- that I might be required to be the  
13 30(b)(6) witness. But as far as being a  
14 spokesperson, I don't know that that was -- I  
09:19 15 guess I don't really understand what you're  
16 saying. It was not -- I didn't have the  
17 understanding that I was going to be doing  
18 public relations work. Is that what you're  
19 referring to or --

09:20 20 Q. Well, let me ask, was there a  
21 separate public relations department?

22 A. Reynolds has a public relations  
23 department, yes.

09:20 24 Q. And does the public relations  
25 department -- is one of their jobs to

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09:20 1 formulate and announce public -- publicly the  
2 company's position on smoking and health  
3 issues?

4 A. They certainly make statements on  
09:20 5 behalf of the company in response to maybe  
6 some scientific allegations, some legal  
7 allegation.

8 Q. Uh-huh.

9 A. They certainly do that, yes.

09:20 10 Q. In doing that, the public  
11 relations department, though, would rely upon,  
12 I assume, your expertise, since you're the  
13 person or were the person most knowledgeable  
14 in those areas?

09:21 15 A. They may call and ask me questions  
16 about an issue, yes, that's true.

17 Q. Were you ever asked during the  
18 period of time that you were director of  
19 smoking and health to formulate a position  
09:21 20 statement, to draft a position statement or to  
21 make some sort of public statement about  
22 smoking and health issues?

23 A. I participated in reviewing the  
24 company's position on -- on smoking and health  
09:21 25 issues, if that's what you're -- you're

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00:21 1 talking about.

2 Q. And were you -- in reviewing that,  
3 were you part of a group or part of a process  
4 that formulated those positions?

09:21 5 A. I had a say in it, yes. I  
6 certainly did.

7 Q. Before you mentioned in your  
8 answer that you might be asked to be the  
9 30(b)(6) representative, you're referring to  
09:22 10 being designated as a corporate representative  
11 to speak on specific issues?

12 A. What -- I'm not a lawyer.

13 Q. Okay.

14 A. This is a term that I have heard  
09:22 15 from lawyers, okay?

16 Q. That's why I asked. It's a very  
17 specific term.

18 A. It is my understanding that that  
19 is a -- a person who is an expert in a certain  
09:22 20 area. That's my understanding of it. And I  
21 have heard the phrase "30(b)(6) witness." But  
22 I have not read the code. I don't know -- I  
23 haven't seen the definition of it.

24 Q. Okay. In making that reference,  
00:22 25 did you have specific instances in mind when

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09:22 1 you were asked to be the company  
2 representative?

3 A. I have given depositions in the  
4 past, yes, as a 30(b)(6) witness.

09:22 5 Q. And what issues, what topics were  
6 you offered as the company's expert of the  
7 person with the most knowledge of?

8 A. I believe it has been a lung  
9 cancer case, peripheral vascular disease. And  
09:23 10 those are the ones that I recall.

11 Q. Okay. So in the lung cancer case,  
12 what was the issue that you were asked to  
13 address by your employer, Philip Morris --  
14 RJR?

09:23 15 A. I don't recall the details. It  
16 just had to do with the overall issue of lung  
17 cancer. As lung cancer has been around for an  
18 awfully long time, our knowledge of lung  
19 cancer and its -- the risk factors for lung  
09:23 20 cancer has evolved rather dramatically over  
21 the last 50 years. We have been able to  
22 identify a number of different risk factors  
23 for it. It's along those lines that --

24 Q. Okay. At some point after 1986,  
09:24 25 you changed positions. Are you currently

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00:24 1 director of smoking and health?

2 A. My title changed to principal  
3 scientist. I still have -- the job  
4 description hasn't changed, but the title has  
09:24 5 changed.

6 Q. Okay. So you've been doing the  
7 same job since 1986?

8 A. Since April of 1986, yes.

9 Q. When did the title change?

09:24 10 A. When I was promoted to principal  
11 scientist. And, now, that's been three or  
12 four years, I believe.

13 Q. Okay.

14 A. I don't recall the date. I'm  
09:25 15 sorry.

16 Q. Do you recall the reason for the  
17 change in job title?

18 A. Well, principal scientist is a --  
19 is a higher -- it represents a promotion.

09:25 20 Q. Uh-huh.

21 A. And so that -- I mean, I was  
22 pleased to be promoted.

23 Q. Was there previously somebody who  
24 was principal scientist above you when you  
00:25 25 were director of smoking and health?

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09:25 1 A. I'm sorry.

2 Q. Were you promoted into an existing  
3 position? Was there a previous principal  
4 scientist who left and you were promoted to  
09:25 5 take his or her place, or was it a new  
6 position that was created?

7 A. There was a person that had the  
8 job director of smoking and health before  
9 me --

09:25 10 Q. Okay.

11 A. -- yes, but he was not a principal  
12 scientist.

13 Q. Were there principal scientists --

14 A. And I don't remember what his  
09:26 15 title was. I'm sorry.

16 Q. I'm sorry. I didn't mean to  
17 interrupt. Was there a principal scientist  
18 before you?

19 A. There are several people within  
09:26 20 R & D who have the title principal scientist.  
21 It is -- it is a level.

22 Q. Okay. How many?

23 A. Oh, three or four. I don't know  
24 the exact number. I'm sorry.

09:26 25 Q. Are the principal scientists

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00:26 1 divided up by subject matter or topic?

2 A. Just give me a second to think how  
3 this is structured. I don't know the exact  
4 structure. But the ones that I am thinking of  
09:26 5 are chemists and work in product development,  
6 as I recall. But I -- I don't know the exact  
7 structure. I'm sorry.

8 Q. Okay. Well, as -- in your  
9 capacity as principal scientist, what is the  
09:27 10 scope of the issues that you are responsible  
11 for?

12 A. The -- the mission is very broad.  
13 One, I have to stay abreast of the -- of the  
14 scientific literature, both physiological,  
09:27 15 biochemical, epidemiological, that would be  
16 related to the field of smoking and health  
17 controversy, smoking and health area. That  
18 means that there are a number of journals that  
19 I have to follow, a number of disciplines that  
09:27 20 I look into.

21 Q. Okay. So you are a principal  
22 scientist in the area of smoking and health?

23 A. That's correct.

24 Q. What areas are not within your  
00:28 25 bailiwick? Is there a -- can you give me a

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09:28 1 general description of things that don't fall  
2 within your area of responsibility?

3 A. Do you mean within the entire  
4 R & D department?

09:28 5 Q. Uh-huh, just some examples.

6 A. Well, in the area of product  
7 development, I mean, I don't have  
8 responsibility for, for instance, cigarette  
9 design or blending or purchasing or analytical  
09:28 10 chemistry. I mean, I know something about  
11 these areas. I interact with the people in  
12 those areas.

13 Q. Uh-huh.

14 A. But I guess -- I'm not sure I  
09:29 15 really understand the question. I would like  
16 to try to give you the information you're  
17 looking for, but I don't know what you're  
18 looking for.

19 Q. With regard to product formulation  
09:29 20 issues, additives, flavorings, specific issues  
21 like that, are you the person with the most  
22 knowledge? Do you have the expertise?

23 A. No, no. Now I understand. All  
24 right. There would be -- in the area of  
09:29 25 cigarette design and product development,

00:29 1 there would be Dr. David Townsend, for  
2 example, would be an expert in that area.

3 Q. And you're not?

4 A. I'm not an expert in that area,  
09:30 5 no.

6 Q. Okay.

7 A. That's not to say I don't know  
8 something about the area. But if you are  
9 looking for an expert, I mean --

09:30 10 Q. Uh-huh.

11 A. -- Dr. Townsend would be the  
12 person. Then there are other people, if you  
13 wanted somebody in analytical chemistry, you  
14 would have to go to someone else. If you  
09:30 15 wanted someone who was an expert on addition,  
16 you would have to go to someone else. I mean,  
17 I might know a little bit about each of these  
18 fields, but I'm not an expert on those fields.

19 Q. Okay. And we've kind of touched  
09:30 20 on it, but do you recall the names of the  
21 other principal scientists at RJR today?

22 A. At one time, Dr. Townsend was, but  
23 he's been promoted to vice president.

24 Q. Okay.

09:31 25 A. I believe Dr. Charles Green is a

09:31 1 principal scientist.

2 Q. What is his area?

3 A. And he is a chemist. I'm sorry,  
4 that's not something I was prepared to answer,

09:31 5 and I don't recall the others who are at that  
6 level. I know that there are some more out  
7 there who are at that level, but it would  
8 be --

9 Q. Okay.

09:31 10 A. -- I would be guessing to -- I  
11 don't know.

12 Q. But the principal scientists in  
13 your organization don't get together  
14 periodically to discuss company policy,  
09:31 15 scientific issues? When was the last time you  
16 met with the other principal scientists at  
17 RJR?

18 A. I might have met with them over  
19 any number of issues and not -- just not  
09:31 20 knowing what their ranks are. I mean, I have  
21 known these people for a long time and I have  
22 been here, as I said, nearly 13 years and  
23 promotions have occurred. And some of them  
24 may be principal scientists and I can't -- and  
09:32 25 I don't know that they have been promoted to

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08:32 1 principal scientists. But I meet with -- I  
2 might meet with a lot of scientists over an  
3 issue of flavor development. I might meet  
4 with them over a blending issue or to discuss  
09:32 5 any number of scientific issues. But it would  
6 be in the nature of product development and  
7 product understanding, for example.

8 Q. And why would you -- why would the  
9 people who were working on flavorings or  
09:32 10 blendings call you into meet with them, what  
11 issues?

12 A. For one thing, I do some work in  
13 the -- in the area of product development and  
14 have some understanding of it. I am a  
09:33 15 chemist, actually a biochemist by education.

16 Q. Uh-huh.

17 A. And have had experience in these  
18 various areas. And that is experience in the  
19 area of inhalation toxicology, air pollution,  
09:33 20 epidemiology and so forth. So I have a wide  
21 range of experience that might be of some  
22 value in a discussion.

23 Q. But if I understand your testimony  
24 correctly, it's not your understanding that  
09:33 25 today, as we sit here today, it is part of

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09:33 1 your job description to be the company  
2 spokesman on smoking and health issues?

3 A. You're using the word "spokesman,"  
4 and I don't consider myself to be a

09:34 5 spokesman. Now, I do consider myself to be  
6 expert in the area of smoking and health. I  
7 spent a lot of time reading the literature, .  
8 studying the literature. But I don't see  
9 myself as a spokesperson for smoking and  
09:34 10 health. If the -- if an occasion requires it,  
11 I expect to be designated as a witness because  
12 of that expertise. But to be just a PR  
13 spokesperson, I don't -- that's not a word  
14 that I would associate with.

09:34 15 Q. All right. We discussed briefly  
16 that you had given some previous testimony.  
17 You've given depositions; is that correct?

18 A. I have, yes.

19 Q. How many depositions?

09:34 20 A. I was asked that question the  
21 other day, and I -- my recollection is I  
22 believe I have given three or four depositions  
23 before. But that's all I can recall. I  
24 didn't make a schedule of it or record of it.

09:35 25 Q. And do you recall the names of the

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0 35 1 cases?

2 A. I was deposed in the Kooper case  
3 in Belleville, Illinois -- and that's spelled  
4 with a K. And then there was a case in  
09:35 5 Kansas, a case in Florida that I was deposed  
6 in. And those are the ones that I recall.

7 Q. Okay. And you've also given  
8 testimony actually at trial, correct?

9 A. I gave testimony at trial in the  
09:35 10 Kooper case in Belleville, Illinois, yes.

11 Q. In each of those instances, in  
12 what capacity were you testifying?

13 A. I believe in all -- well, I don't  
14 recall. In the Kooper case -- I do not  
09:36 15 believe I was a 30(b)(6) witness in the Kooper  
16 case. I just don't recall. It's been several  
17 years ago. But the others I might have been a  
18 30(b)(6) witness, I don't know. I don't  
19 recall.

09:36 20 Q. Okay. As part of your preparation  
21 for your testimony today, did you review your  
22 previous testimony?

23 A. I'm trying to remember. I haven't  
24 reviewed it recently. I haven't looked at it  
09:36 25 recently. So I guess you could say that I did

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09:36 1 not for this particular --

2 Q. All right. What things did you do  
3 to prepare for your deposition today?

4 A. Well, I looked at the -- for one  
09:37 5 thing, there's not a whole lot that you can  
6 really do to get ready for a deposition,  
7 okay? I look at the fairly recent papers and  
8 then at papers that might be review-type  
9 articles that would be broad spectrum in their  
09:37 10 analysis of smoking and health issues.

11 But frankly, not a lot.

12 Q. Okay.

13 A. I mean, I didn't spend a lot of  
14 time preparing for it.

09:37 15 Q. Did you review any -- any  
16 documents, any correspondence from your files  
17 or from the company's files?

18 A. Not that I recall. I don't recall  
19 doing any of that, no.

09:37 20 Q. Okay. So the only thing -- if I  
21 understand what you're saying correctly, the  
22 only thing you've reviewed is some of the more  
23 recent literature that would fall under the  
24 category that we've discussed previously,  
09:38 25 which is --

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00:38 1 A. Well, I might look back at  
2 something that was a big document.

3 Q. Uh-huh.

4 A. And it might not be very recent.  
09:38 5 Let me see if I can explain this to you. A  
6 document might be published in the early 1980s  
7 that was a substantial -- a huge review that  
8 was very interesting written by people who are  
9 very prominent in the field. No matter  
09:38 10 whether they are good or not, they are very  
11 prominent and they have done a massive job of  
12 reviewing an article. Okay. So it stands in  
13 1981. Well, many years later, say, 15,  
14 20 years later, somebody may revisit that  
09:38 15 review.

16 Q. Okay.

17 A. May take a section of it, revisit  
18 it. And it reopens curiosity, it reopens  
19 interest. What has changed about the review  
09:39 20 that was done in 1980 or '81, another one is  
21 being done today and is going to be on the  
22 table. How has the position changed. Is  
23 there new information? Is there a new view or  
24 interpretation of that information? It's that  
00:39 25 sort of thing that -- that I would be

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09:39 1 curious. And, I mean, I see this as part of  
2 my responsibility, not only how is the  
3 evidence changing with time, how is the view  
4 of that evidence changing with time. Is

09:39 5 that --

6 Q. Uh-huh.

7 A. I'm not making myself clear, but  
8 that's roughly what I'm --

9 Q. That's fine. Are there specific  
09:39 10 studies, collections of data that you're  
11 thinking of that you've reviewed in  
12 preparation for this deposition or maybe  
13 recently in preparation generally for this  
14 case and others?

09:40 15 A. The specific case that I was  
16 thinking about when I was trying to give you  
17 that example, remember, my job is -- is to try  
18 to keep from going blind from reading too  
19 much.

09:40 20 Q. Uh-huh.

21 A. I mean, I read an awful lot. I  
22 have -- there are a lot of papers that come  
23 across my desk and I have to study these  
24 papers and try to understand them as much as I  
09:40 25 can in the context of the -- of the field,

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09:40 1 might be a biochemical field, it might be  
2 epidemiology or something else. But the one I  
3 was thinking about which is fascinating which  
4 I want to spend some more time on, in the  
09:40 5 early '80s, Sir Richard Dahl and a man named  
6 Richard Peto published a pretty massive review  
7 of cancer in the United States. Now, both of  
8 these gentlemen are from the UK. And it was  
9 kind of interesting the way they partitioned  
09:41 10 cancer in the United States between nutrition,  
11 tobacco, and other life-style factors, as I  
12 recall.

13 Now, this is interesting. That's  
14 16 years ago. Well, I have just received  
09:41 15 information that two new documents are going  
16 to be coming out, one from the United Kingdom  
17 and one from an arm of the world health  
18 organization in which they are ascribing an  
19 awful lot -- a high percentage of cancer to  
09:41 20 nutrition.

21 Now, not having seen those  
22 documents, I wanted to get an idea just, okay,  
23 what has been the thinking up to this time,  
24 because the -- the report that I got was --  
09:42 25 and I haven't seen the documents -- that they

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09:42 1 were ascribing an awfully high percentage of  
2 cancer to nutritional factors, much higher  
3 than I had seen in the past. In the past I  
4 had seen figures between 30 and 40 percent  
09:42 5 could be ascribed to some sort of nutritional  
6 factor.

7 But the article that I read .  
8 indicates that these two new papers, which are  
9 very much in agreement, will be between 60 and  
09:42 10 70 percent.

11 Now, this is a pretty substantial  
12 change over the review done by Dahl and Peto  
13 back in the early '80s. And I'm curious about  
14 this. I would like to know what evidence they  
09:42 15 are using. So when I saw the blurb, I made  
16 efforts to try to get a copy of it, but they  
17 are not going to be available until early  
18 September, so I haven't had a chance to look  
19 at them, but I wanted to go back through the  
09:43 20 nutritional information anyhow.

21 Q. And when you say the blurb, you  
22 saw an abstract somewhere?

23 A. Exactly, and I'm trying to  
24 remember where I saw it. But it was specific  
09:43 25 enough for me to know who to contact, or at

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00:43 1 least for our librarian to know who to contact  
2 to try to get a copy of the document. And she  
3 found out that it would not be available  
4 until -- it's in draft form at the moment and  
09:43 5 will not be available for distribution until  
6 early September or sometime early in  
7 September.

8 Q. You've mentioned two specific  
9 documents, and just so that I don't get  
09:43 10 confused, let's talk about them one at a time  
11 so you can tell me --

12 A. Okay.

13 Q. First one, pick one and tell me  
14 who -- what your understanding of who's  
09:44 15 writing the article and where it's coming  
16 from.

17 A. Okay. You're referring now to the  
18 new documents --

19 Q. Yes, sir.

09:44 20 A. -- that I have seen a blurb on?

21 Q. Yes, sir.

22 A. Okay. This document, the one I  
23 thought would be available because a draft has  
24 already been released on this document, I just  
00:44 25 can't get ahold of it, is coming out of the

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09:44 1 United Kingdom and it's written by -- I  
2 believe the health ministry is responsible for  
3 that, putting that document out.

4 Q. It's a government publication?

09:44 5 A. And this is a government  
6 publication.

7 Q. Do you know what the title is or.  
8 the key words or anything like that?

9 A. Well, "nutrition" is the key word.

09:44 10 Q. Okay.

11 A. I think nutrition is the one you  
12 want to look for. I think that's the --

13 Q. And I'll bet "smoking" is the  
14 other key word?

09:44 15 A. I don't know whether they deal  
16 with smoking in that one or not.

17 Q. Okay.

18 A. I think what they're focusing on  
19 is nutritional practices. And there's a

09:44 20 reason for that. In the UK, the fruits and  
21 vegetables are not consumed as regularly as  
22 they are in -- in a lot of other countries, in  
23 the United States, for example. Scotland is  
24 really tough. They consume a lot of animal  
09:45 25 fats. They don't get a lot of fruit. I mean,

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00:45 1 bananas are an endangered species in  
2 Scotland. I was over there this year. And  
3 you don't just find fruit commonly.

4 Q. Uh-huh.

09:45 5 A. And they have interesting rates  
6 of -- or disturbing rates of cardiovascular  
7 disease, lung cancer, which -- and other types  
8 of cancer which can be linked, apparently, to  
9 nutrition.

09:46 10 Now, a few years ago, a conference  
11 was held at the University of Kentucky on  
12 smoking and nutrition. And people came from  
13 all over the world to attend this conference.  
14 I happened to be there, and you had people  
09:46 15 from Scotland. You had people from Canada and  
16 Germany and France and so forth that were  
17 looking at this particular issue, the  
18 nutritional aspect of cancer --

19 Q. Uh-huh.

09:46 20 A. -- and cardiovascular disease.  
21 And one of the things that came out of it was  
22 nutrition -- that I got out of it, there were  
23 a lot of very interesting conferences -- was  
24 the nutritional practices around -- in the  
00:46 25 western world.

51710 1097

09:46 1 Anyhow, because of that, I'm  
2 interested in these two documents. The first  
3 one was the UK document that you asked me  
4 about. The second one is this document that's  
09:47 5 being put out from a branch -- my recollection  
6 is it is a branch of the World Health  
7 Organization. It's the world -- I can't  
8 remember the exact title of it. But it's --  
9 it's a very different document from the UK.

09:47 10 Q. Uh-huh.

11 A. Now, according to the blurb that I  
12 saw, their conclusions are going to be  
13 similar, but they're prepared by two different  
14 groups. And, of course, because of the  
09:47 15 earlier review that I had read by Dahl and so  
16 forth, I'm very interested in this and also  
17 because of that smoking and nutrition  
18 conference that was held in Kentucky.

19 Q. On the World Health Organization  
09:48 20 publication, do you recall what population  
21 group that that study addressed?

22 A. I do not believe that that  
23 information was included in the blurb. It was  
24 just -- it dealt -- both articles dealt with a  
09:48 25 high percentage of cancer that can be

51710 1098

09:48 1 attributed to nutrition. And it was well  
2 above the percentages that Dr. Dahl had  
3 referred to in his earlier review, above  
4 60 percent, between 60 and 70 percent, which  
09:48 5 is very different from what you've seen  
6 before. Most other reports that I've seen  
7 have been in the 30 to 35 percent range. And  
8 so these are high. And I don't -- I would  
9 like to know why they are high, what they base  
09:49 10 that conclusion on.

11 Q. And we are talking 60 to  
12 70 percent of all cancers?

13 A. This is all cancers, is my  
14 understanding. I haven't seen the documents,  
09:49 15 but that's my understanding.

16 Q. Okay. Just one follow-up. The  
17 conference you referred to -- this is smoking  
18 and nutrition -- were the proceedings  
19 published?

09:49 20 A. They were published as a New York  
21 Academy of Sciences monograph, as a matter of  
22 fact. And even though those things are fairly  
23 expensive, it is on the market, can be  
24 purchased, yes.

09:49 25 Q. Okay. Any other specific issues

51710 1099

09:49 1 that come to mind that you've thought about,  
2 examined recently in preparation for your  
3 testimony?

4 A. I'm sorry, I can't -- I don't  
09:50 5 recall whether there were or not. I don't  
6 recall any. I mean, I may have. I didn't  
7 make a huge issue out of getting ready for the  
8 deposition, so I -- I don't recall.

9 Q. Have you created any documents in  
09:50 10 preparation for your testimony in this case?

11 A. There was, I guess, the one  
12 document I participated in creating was the  
13 list of opinions that was submitted.

14 MR. THOMPSON: Just for the  
09:50 15 record, I will go ahead and mark as Exhibit 1  
16 as --

17 (Deposition Exhibit 1  
18 was marked.)

19 Q. (By Mr. Thompson) Is that the  
09:50 20 list of opinions you are referring to, Doctor?

21 A. Let me just take a quick look.  
22 This is it.

23 Q. Okay. Did you prepare the  
24 document and forward it to the attorneys?

09:51 25 A. This document was prepared after

51710 1100

00:51 1 conversations that I've had with our defense  
2 counsel over the years. It's consistent with  
3 discussions that I've had. It was not -- I  
4 did not write it.

09:51 5 Q. Okay.

6 A. But it was submitted to me for my  
7 approval.

8 Q. So it was written by, I guess, the  
9 lawyers?

09:51 10 A. I don't know who wrote it.

11 Q. Okay.

12 A. But it was submitted to me, and I  
13 was asked, is this consistent with your views  
14 of your job. And that's correct.

09:51 15 Q. Okay. And when you say it was  
16 submitted to you, who submitted it to you?

17 A. I believe that I got a fax copy  
18 from this lady right here.

19 Q. Okay. Did you make any changes to  
09:52 20 the draft?

21 A. I don't recall that I had any  
22 changes to make at all.

23 Q. Okay. When you say it was  
24 consistent with your recollection or your view  
00:52 25 of your job, is this a document that you

51710 1101

09:52 1 previously were involved directly in creating  
2 and just another copy of it or --

3 A. For example, that's the only way I  
4 know to answer your question.

09:52 5 Q. That's fine.

6 A. In Item 4, expected to testify  
7 that the mechanism or mechanisms of the  
8 induction of chronic, noninfectious, lung,  
9 latent period diseases such as --

09:52 10 MS. FORBES: Slow down. It's  
11 just a little easier for the court reporter.

12 THE REPORTER: Thank you.

13 A. Sorry. It's Item 4. This is a  
14 result of discussions that we've had over --  
09:53 15 well, since 1986, actually '87 I spent a lot  
16 of time studying the literature after about a  
17 year before I took the job, and then after I  
18 took the job. And so I've had discussions  
19 with legal counsel over these issues for a  
09:53 20 number of years. So it's not something that  
21 was prepared on the spur of the moment, so to  
22 speak.

23 Q. (By Mr. Thompson) Right. But  
24 this is -- these statements and you use the  
09:53 25 example, I think, of -- did you say Paragraph

51710 1102

00:53 1 4?

2 A. Number 4, Item Number 4.

3 Q. -- are opinions that have been  
4 developed over time, if I understand your  
09:53 5 testimony correctly, through conversations  
6 between you and counsel?

7 A. Actually, they're opinions that I  
8 had in one form or another based on review of  
9 the literature, based on conversations with  
09:53 10 people, just based on general experience, I  
11 guess, through my career for that particular  
12 one. We don't know a mechanism, for example,  
13 by which -- a biochemical mechanism by which  
14 cancer is initiated and propagated.

09:54 15 Q. The opinions set out in Exhibit 1,  
16 have they been the same -- you've said that  
17 this is a process began in 1986. Have any of  
18 these points substantially changed over time?  
19 Have you asked your lawyers to make changes to  
09:54 20 those statements?

21 A. Opinions, scientific opinions are  
22 always the result of some evolution, process  
23 of evolution. As evidence changes or as  
24 theories change, opinions can change. And I  
00:55 25 don't have all of these before me to see how

51710 1103

09:55 1 they might have evolved. But I'm sure that if  
2 you went back through, you might find  
3 differences between the different ones that  
4 have been submitted. I don't know. I didn't  
09:55 5 go back and look.

6 Substantively, the evidence has  
7 not really changed in this area as far as the  
8 type of evidence that you have. You have, as  
9 I point out -- pointed out here in Item 1,  
09:56 10 epidemiology, toxicology, chemistry, and  
11 mechanism. These factors have been pretty  
12 much the same for a long period of time,  
13 epidemiology has been positive, the animal  
14 studies have been negative. We still don't  
09:56 15 know a mechanism.

16 Theories have changed. We have  
17 seen some interesting evolution in theories  
18 with time. Some have been tested, found to be  
19 inadequate and discarded. But this is what  
09:56 20 you do. You don't discard evidence. You  
21 discard theories.

22 Evidence represents reality,  
23 theories represent somebody's imagination,  
24 which is powerful, needs to be there. But  
09:56 25 there's an old adage in science that goes back

51710 1104

00:56 1 to Max Plunk, when the two are in conflict,  
2 experimental fact has sovereignty over  
3 theoretical subtlety. No matter how elegant  
4 your theory may be, if I won't explain the  
09:57 5 facts, it has to go. You have to come up with  
6 a new theory.

7 But -- so theories have evolved:  
8 The evidence has pretty much stayed the same.  
9 There's more of it, but it's of the same  
09:57 10 theme.

11 Q. And if I understand what you said  
12 correctly, the -- it's the evidence, it's the  
13 underlying facts that really are the most  
14 important thing in a scientific inquiry.

09:57 15 A. The theory is always important,  
16 don't misunderstood what I'm saying, but the  
17 evidence -- the valid data have sovereignty  
18 over the theory. And you use experimentation  
19 to test theory.

09:57 20 If the theory will not encompass  
21 and explain the evidence, then the theory has  
22 to go, not the evidence.

23 Q. All right. Have you been asked to  
24 prepare any documents or exhibits for use at  
09:58 25 trial in this case.

51710 1105

09:58 1 A. In this case?

2 Q. Uh-huh.

3 A. No, sir, I have not, not for this  
4 case. I have participated in the preparation  
09:58 5 of exhibits for other cases, but not this one.

6 Q. Okay. Have you specifically been  
7 asked to appear at trial in this case?

8 A. I don't think so. I have a -- not  
9 that I recall.

09:58 10 Q. Okay. Do you know when the trial  
11 is?

12 A. No, sir, I don't.

13 Q. What is your understanding of the  
14 capacity that you've been asked to appear in  
09:59 15 this case, in the State of Texas case?

16 A. Mr. Thompson, I know so little  
17 about legal proceedings and about --  
18 particularly -- as I understand it, this is an  
19 attorney called -- referred to as an attorney  
09:59 20 general's case, is that --

21 Q. Uh-huh.

22 A. -- correct?

23 Frankly, I'm not sure what  
24 capacity I would be called in. I just don't  
09:59 25 understand the case.

51710 1106

0 59 1 Q. Okay. Is it your understanding  
2 you've been asked to give testimony as  
3 spokesman for RJR on health -- smoking and  
4 health issues?

09:59 5 A. Not as spokesman. I haven't heard  
6 that. I was told to show up here today for  
7 deposition. I don't consider this being a  
8 spokesperson for RJR. This is a -- I'm  
9 appearing to give my opinions on different  
10:00 10 subjects.

11 Q. Okay. And is it -- you've been  
12 asked to appear as an expert witness; is that  
13 your understanding?

14 A. Exactly, yes, sir, that's correct.

10:00 15 Q. Okay. And the area that you've  
16 been asked to give your opinions in is the  
17 area of smoking and health?

18 A. That's correct.

19 Q. Is that a -- an area that is a  
10:00 20 discipline, a scientific discipline?

21 A. Let's go back to -- in order to  
22 answer that, go back to Item 1.

23 Q. Okay.

24 A. Look at the lines of scientific  
10:00 25 evidence that appear there. Epidemiology is a

51710 1107

10:01 1 discipline in itself. Toxicology is a  
2 discipline in itself. Chemistry is a separate  
3 discipline. And mechanism refers really to  
4 biochemistry, and biochemistry would be sort  
10:01 5 of a subset of chemistry. Biochemistry is a  
6 study of those chemical reactions involved in  
7 a life process, a living process. So you have  
8 four different disciplines that you're -- that  
9 you're looking at.

10:01 10 So to say that smoking and health  
11 is a -- is one area is probably a little too  
12 confining. It really branches out and it has  
13 to touch a lot of things.

14 Q. Uh-huh.

10:01 15 A. And there's a lot of literature to  
16 review.

17 Q. Understood. And let's use your  
18 Paragraph 1, I guess, as a starting point.  
19 Number 1, you've listed epidemiology as one of  
10:02 20 the areas. Are you an epidemiologist?

21 A. No, sir, I'm not.

22 Q. Do you have any formal training in  
23 epidemiology?

24 A. I have never taken a formal course  
10:02 25 in epidemiology, no, sir.

51710 1108

02 1 Q. Okay. Are you a toxicologist?  
2 A. No, I have never taken a formal  
3 course in toxicology.  
4 Q. Okay. So you wouldn't hold  
10:02 5 yourself out to be an expert in epidemiology?  
6 A. No.  
7 Q. Or in toxicology?  
8 A. No.  
9 Q. You do have a chemistry  
10:02 10 background?  
11 A. I have a Ph.D. in biochemistry,  
12 yes.  
13 Q. So -- and that is different --  
14 that would not apply to Number 3, which is  
10:02 15 chemistry?  
16 A. As a matter of fact, in order to  
17 receive a degree in biochemistry, you have to  
18 take an awful lot of chemistry courses,  
19 physical chemistry, organic chemistry,  
10:03 20 analytical chemistry.  
21 Q. Uh-huh.  
22 A. And understand, because in  
23 biochemistry you use all of these various  
24 aspects. Toxicology is a very specific  
10:03 25 discipline within a broader area that you

51710 1109

10:03 1 might define as pharmacology.

2 Q. Okay. And you're not a  
3 pharmacologist?

4 A. No, I'm not a pharmacologist.

10:03 5 Q. Have you had any formal training  
6 in pharmacology?

7 A. I have actually taught aspects of  
8 pharmacology.

9 Q. Okay.

10:03 10 A. I was at the Milton S. Hershey  
11 Medical Center for a year and a half. And  
12 using biochemistry, my biochemistry background  
13 and the research that I was doing, I taught  
14 some aspects of pharmacology.

10:04 15 Q. And when you say "some aspects,"  
16 you mean to the extent biochemistry is a  
17 component of that bigger discipline, you were  
18 involved in the biochemistry part of it?

19 A. A little different from that.

10:04 20 Q. Okay.

21 A. The -- the course that is taught  
22 generally to medical students is taught by  
23 five or six different people. One person is  
24 not charged with -- or at least in this

10:04 25 instant was not -- instance was not charged

51710 1110

10:04 1 with the responsibility of teaching the entire  
2 pharmacology -- you have pharmacologists who  
3 might be specialists in central nervous system  
4 pharmacology. Another might be a specialist  
10:04 5 in cardiovascular pharmacology. And so they  
6 would teach the areas in which they were  
7 expert in.

8 Q. Uh-huh.

9 A. Now, the area that I was expert in  
10:05 10 was cardiovascular. I was -- had fairly  
11 extensive knowledge at one time of a class of  
12 drugs that are referred to as cardiac  
13 glycosides used to treat congestive heart  
14 failure. A very important class, a very  
10:05 15 dangerous class of drugs. And it was in this  
16 area that I taught.

17 Q. So the teaching that you referred  
18 to is in this very narrow area of pharmacology  
19 as it relates to specific, I guess, drugs that  
10:05 20 relate to treatment of --

21 A. Yes.

22 Q. -- of congestive heart failure?

23 A. Remember, there are a lot of  
24 peripheral issues that surround something like  
10:05 25 that. For example, if I want to talk about

51710 1111

10:06 1 the efficacy of cardiac glycoside, that's one  
2 thing. But you don't just teach a medical  
3 student the efficacy of the drug. You have to  
4 talk about the dose relationship. You have to  
10:06 5 talk about the adverse effects, potential  
6 adverse effects, how do you treat the  
7 potential adverse effects.

8 So really you get into the  
9 pharmacology of the drug, which would be  
10:06 10 the -- the pharmacokinetics and  
11 pharmacodynamics of the drug. Then you get  
12 into the toxicology of the drug. And from  
13 there, you get into the treatment of these  
14 adverse effects, which is another form of  
10:06 15 pharmacology. For instance, cardiac  
16 glycosides are very prone to throw  
17 arrhythmias. Some of these can be very  
18 dangerous.

19 Q. Kind of defeats the purpose if it  
10:06 20 causes the heart to stop working?

21 A. Well, the idea is that in  
22 congestive heart failure, the heart is just  
23 not putting out enough blood.

24 Q. What causes congestive heart  
10:07 25 failure?

51710 1112

10:07 1 A. It is very much an inherited  
2 disease. Now, I can't tell you what  
3 percentage would be attributed to genetics,  
4 what might be attributed to something else,  
10:07 5 but I know it's not all genetics. But you  
6 find a very strong vein of this genetic  
7 effect.

8 And the cardiac glycosides have  
9 this very pronounced ability to modify that so  
10:07 10 that the heart puts out a lot more blood. And  
11 therefore, the -- your edema goes down. But  
12 that can cause an arrhythmia and interfere with  
13 the electrical conduction of the heart. So  
14 then you have to -- when you're teaching it,  
10:07 15 then you have to talk about, okay, you're a  
16 physician, you've given cardiac glycoside to  
17 somebody and it looks like it's going well and  
18 all of a sudden he's got an arrhythmia, what  
19 do you do. And so there's several drugs that  
10:08 20 you give to try to offset the arrhythmia. So  
21 it becomes -- it's not a simple issue when you  
22 begin to try to teach. You can't really  
23 isolate it that well.

24 MS. FORBES: Jeff, if this is  
10:08 25 a good place, could we just take a quick --

51710 1113

10:08 1 MR. THOMPSON: Let me ask one  
2 question and it would be a great place to  
3 break.

4 Q. (By Mr. Thompson) Do cardiac  
10:08 5 glycosides have anything to do with smoking  
6 and health?

7 A. Right offhand, I don't know. I  
8 don't -- I don't see the connection.

9 MR. THOMPSON: Okay. Great  
10:08 10 place for a break.

11 THE VIDEOGRAPHER: Off the  
12 video record.

13 (A recess was taken.)

14 THE VIDEOGRAPHER: We're on  
10:21 15 the video record.

16 Q. (By Mr. Thompson) Doctor, we're  
17 back, we took a short break. We were talking  
18 about your capacity as an expert in smoking  
19 and health in this case.

10:22 20 A. Yes.

21 Q. And we had gone through some of  
22 the different areas that you've -- you believe  
23 are involved in that bigger subject. Let me,  
24 if I could, just talk with you a minute about  
10:22 25 your background. You, in 1958, received a BS

51710 1114

10:22 1 in chemistry. Was it from Auburn or from the  
2 Alabama Polytechnic Institute?

3 A. At that time was Alabama  
4 Polytechnic Institute.

10:22 5 Q. It's become Auburn since that?

6 A. And I don't recall the exact date,  
7 but it was sometime in the '60s that I guess  
8 the university officials decided nobody ever  
9 called it Alabama Polytechnic Institute. It

10:23 10 was always known as Auburn, so they changed  
11 the name to Auburn University in 19 -- or in  
12 the 1960s, and I don't know. And so I have  
13 always listed because my bachelor's degree  
14 reads Alabama Polytechnic Institute and my  
10:23 15 Ph.D. reads Auburn University.

16 Q. Did -- at that time, in your  
17 undergraduate studies, did you take any  
18 courses, do any specific studies in the area  
19 of smoking and health?

10:23 20 A. No, sir.

21 Q. Did you take any courses or do any  
22 specific work in the area of epidemiology?

23 A. No, sir, I did not.

24 Q. Did you do any specific course  
10:23 25 work or studies in the area of toxicology?

51710 1115

10:23 1 A. I took some courses that were  
2 peripheral to toxicology. They were taught in  
3 the animal science. One was taught in the  
4 animal science -- department of animal  
10:23 5 science, and another was taught over in the  
6 school of veterinary medicine.

7 Q. But you did not specifically study  
8 toxicology or receive a degree in toxicology?

9 A. I did not, no.

10:24 10 Q. You then went on and studied, I  
11 guess, at Auburn, renamed, and received a  
12 Ph.D. in biochemistry in 1968, correct?

13 A. That is correct, yes.

14 Q. In that course program, did any of  
10:24 15 the classroom research work address smoking  
16 and health issues?

17 A. No, they did not.

18 Q. Did you have any course work in  
19 the Ph.D. program in the area of epidemiology?

10:24 20 A. No.

21 Q. Did you have any course work or  
22 work in the Ph.D. program in the area of  
23 toxicology?

24 A. Once again, I took some -- some  
10:24 25 courses in the animal science -- department of

51710 1116

10:24 1 animal science, which related to nutritional  
2 aspects of toxicology, yes. But that's the  
3 extent of that.

4 Q. But no specific course work in  
10:25 5 toxicology?

6 A. No, sir.

7 Q. Have you ever published on the  
8 area of smoking and health?

9 A. No, I have not.

10:25 10 Q. Have you ever published in the  
11 area of epidemiology?

12 A. I have not.

13 Q. Have you ever published in the  
14 area of toxicology?

10:25 15 A. I have not.

16 Q. In fact, you have -- have you been  
17 the principal author in any publication that's  
18 been published in peer review literature?

19 A. A couple of papers. One was on  
10:25 20 cardiac glycosides, and another was on a drug  
21 used to treat meningeal leukemia.

22 Q. And you were the principal author  
23 on those papers?

24 A. I believe that's correct, yes,  
10:26 25 sir.

51710 1117

10:26 1 MS. FORBES: Jeff, maybe if  
2 you put a resume in front of him, it might be  
3 helpful.

4 MR. THOMPSON: I will. It's  
10:26 5 not here.

6 A. Let's see. I believe I was  
7 principal author on Number 1, Effects of  
8 Cardiac Glycoside on Plasma Phospholipid  
9 Levels, and Number 2, Quantitation of Peer  
10:27 10 Methane and Related Diamylenal Pyrimidines and  
11 Chromatography.

12 Q. (By Mr. Thompson) Okay. On the  
13 CV it doesn't list you as principal author; is  
14 that correct?

10:27 15 A. I think that this CV was prepared  
16 to show publications that I was on with other  
17 authors. So it does not show my name  
18 positioned -- I'm on all of these papers.

19 Q. Okay.

10:27 20 A. And --

21 Q. But -- and I don't mean to  
22 interrupt, but it's -- kind of the protocol in  
23 the scientific community is for the principal  
24 author to be listed first; is that correct?

10:27 25 A. That is pretty universal. There

51710 1118

1 are cases where the last author sometimes  
2 might be considered, but not an intermediate  
3 author. In other words, it would be --  
4 99 percent of the time it would be the first  
10:27 5 author. Sometimes it will be the -- for  
6 example, if a guy published with his major  
7 professor -- published a paper with his major  
8 professor.

9 Q. You couldn't put the professor  
10:27 10 first?

11 A. Actually, it doesn't matter where  
12 he comes. He's a principal author.

13 Q. Understood. So on the CV,  
14 Number 1 and 2 on your CV --

10:28 15 A. I believe I'm principal author on  
16 those papers.

17 Q. And those articles were  
18 published -- one was published in the -- what  
19 is that?

10:28 20 A. Research Communications and  
21 Chemical Pathology and Pharmacology.

22 Q. Okay.

23 A. And the second one is in  
24 Analytical Chemistry.

10:28 25 Now, I may have been principal

51710 1119

10:28 1 author on a few of these papers right here,  
2 but I'd have to --

3 MS. FORBES: And just so the  
4 record reflects he is just referring to --

10:28 5 A. 6, 7, and 8. I may have been.  
6 I'd have to go back and look. I don't know if  
7 I could even find copies of those.

8 Q. (By Mr. Thompson) Okay. Do any  
9 of those articles that you're referring to  
10:29 10 that you were principal author on have  
11 anything to do with smoking and health?

12 A. Only in the sense that they deal  
13 with pharmacology, biochemistry, and to a  
14 certain extent, toxicological response, but  
10:29 15 not specifically smoking and health, no.

16 Q. Okay. To the best of your  
17 recollection, are those the only peer review  
18 publications that you were principal author  
19 in?

10:29 20 A. I believe that's correct, yes.

21 Q. And let me ask you to look -- just  
22 take a minute and look at that list, see if  
23 there are any omissions that come to mind.  
24 And I understand that it's hard to -- just if  
10:29 25 you will look at the list and tell me whether

51710 1120

29 1 you believe that's a complete list of your  
2 publications.

3 A. Mr. Thompson, I believe that it  
4 is.

10:30 5 Q. Okay. And it looks to me like  
6 there are some -- there are 22 publications  
7 listed. There are several that look like they  
8 are just duplications.

9 A. On this --

10:30 10 Q. And let me just direct your  
11 attention to Number 19 and Number 21.

12 A. All right. I don't -- I don't  
13 believe those are duplications.

14 Q. Okay.

10:31 15 A. It's been a long time. One of  
16 these reports was done for the Electric Power  
17 and Research Institute in Palo Alto.

18 Q. Okay.

19 A. Another was done for Southern Cal  
10:31 20 Addison, which is located -- it says here  
21 Rosemead, but down in the Los Angeles area.

22 Q. Okay.

23 A. And I don't believe that those are  
24 duplications.

10:31 25 Q. Okay. What about the other

51710 1121

10:31 1 number? And you're referring on those two to  
2 which two numbers?

3 A. You had called my attention to 19  
4 and 21 is what I was talking to, are these  
5 duplications.

6 Q. Right. Is 21 a duplication of  
7 Number 13?

8 A. Okay. Now, that appears to be a  
9 duplication. It has the same -- it has the  
10:32 10 same number on it. In this case, we prepared  
11 several different reports on toxicological  
12 profiles. In other words, the same substances  
13 might not -- would not be in both reports, but  
14 they might have similar titles.

10:32 15 Now, because that number is  
16 similar -- not similar, it's exact, it looks  
17 like it may be a duplication.

18 Q. All right. Just briefly, can you  
19 tell me what -- what this toxicological  
10:32 20 profile entailed, what kind of document was  
21 it?

22 A. The -- once again, this is a --  
23 this is a literature-intensive effort.

24 Q. Okay.

10:32 25 A. And what we were attempting to do

51710 1122

10:32 1 is from published lists of chemicals that had  
2 been identified, associated with some emission  
3 or some process, try to prepare an overview of  
4 what is known about its potential biological  
10:33 5 activity in humans. "Biological activity" is  
6 a really broad term. We might look for almost  
7 any kind of response. Eye watering -- any  
8 kind of response that might have been reported  
9 in the scientific literature. It could refer  
10:33 10 to taste.

11 But primarily, the evidence that  
12 we looked at would have been taken from either  
13 some in vitro study, which were being done  
14 during this period of time, or some animal  
10:34 15 study that had been reported on that chemical  
16 or that mixture of chemicals.

17 Obviously, the reason for this is  
18 that your utility industry was going toward  
19 newer process -- newer combustion processes,  
10:34 20 fluidized bed combustion was coming in, newer  
21 types of emission abatement controls. And  
22 this changes the mix of things that are  
23 emitted into the atmosphere.

24 Q. Okay. Products of combustion?

10:34 25 A. Products of combustion or -- yeah,

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10:34 1 that could be modified by the way you try to  
2 abate -- you change the way you burn things,  
3 you might change the way things go up a stack.

4 Q. Okay.

10:35 5 A. Okay. So there were a lot of  
6 technologies that were being developed. Now,  
7 what they were interested in is how do we  
8 establish strategies for keeping pollutants  
9 down below what might be some -- either some  
10:35 10 state standard or some national standard. You  
11 need evidence. What things need to have a  
12 standard, for example.

13 So what we were doing is trying to  
14 create a picture for our clients that they  
10:35 15 could look at, see -- identify potential  
16 problems so that they could address those  
17 problems. I don't know if that --

18 Q. Uh-huh.

19 A. -- makes any sense to you. But  
10:35 20 it's very literature intensive. We had to go  
21 through an awful lot of peer review literature  
22 to get these types of data.

23 Q. And you gathered the data that was  
24 available about a substance or substances and  
10:35 25 provided that information to your client so

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10:35 1 they could make, I guess, decisions based on  
2 that information?

3 A. Yeah. They -- or -- when you --  
4 "decision" is a pretty broad term. It might  
10:36 5 help them and give some guidance in how they  
6 design a -- a process --

7 Q. Uh-huh.

8 A. -- or something. I mean,  
9 information is -- is very, very important.  
10:36 10 Good information is very important. It does  
11 something else. If I go out and do an  
12 exhaustive review of the literature on a given  
13 subject, one thing that I will identify will  
14 be data gaps. What needs to be known, not  
10:36 15 only what do I know. It raises a question, do  
16 I believe it. What is not known, what other  
17 questions need to be asked. So they can use a  
18 report like that probably in a lot of  
19 different ways. And so the main thing is to  
10:37 20 give them an information profile.

21 Q. What types of substances did you  
22 generally investigate in that profile?

23 A. Most of the substances that would  
24 be associated with the combustion, or a lot of  
10:37 25 substances that would be associated with the

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10:37 1 combustion of fossil fuel, either natural gas  
2 or oil or coal, or it might be involved in the  
3 refining of petroleum or the -- and one of the  
4 issues that was very prominent at that time --  
10:38 5 I wish it had gone on -- was the coal  
6 gasification process. We have enormous coal  
7 reserves in this country, but we have limited  
8 petroleum reserves, or not as extensive as our  
9 coal reserves. But coal can be converted into  
10:38 10 a very good fuel.

11 Q. Uh-huh.

12 A. But it requires a technology and  
13 it involves producing pollution. So how do  
14 you do this in a way that is compatible with  
10:38 15 good environmental and public health  
16 management.

17 Q. And is that a -- an analysis that  
18 a responsible industry should go through?

19 A. I'm not sure I understand that --  
10:38 20 that question. Is it an analysis that an  
21 industry should go through?

22 Q. Uh-huh, in your opinion.

23 A. It is a process that every  
24 industry I've ever been associated with,  
10:38 25 including pharmaceutical industry, goes

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10:38 1 through in one form or the other. They --  
2 they gather information. They gather -- and  
3 evaluate the quality of that information and  
4 try to determine how do I act based on  
10:39 5 information. You don't act in the absence of  
6 information. You take that information, do  
7 the best you can to convert it into something  
8 that is usable, and then you act on it.

9 Did I answer your question?

10:39 10 Q. Why does industry do that?

11 A. Because industry does not act  
12 randomly. People who act randomly go out of  
13 business. You -- you get -- information gives  
14 you guidance in your decisions.

10:39 15 Q. Okay. Look at Number 19 real  
16 quickly, just because we skipped over it. Is  
17 that the same as Number 11?

18 A. Once again, Mr. Thompson, it looks  
19 like a duplicate.

10:40 20 Q. Okay. Tell me what that  
21 publication was about in general.

22 A. In this particular publication we  
23 were looking for -- do you know what the South  
24 Coast Air Basin is?

10:40 25 Q. No idea.

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10:40 1 A. All right. Los Angeles is located  
2 in the South Coast Air Basin of California.  
3 I'm sorry.

4 Q. That's okay. Go ahead.

10:40 5 A. It is referred to as a basin  
6 because of the geographical features. It  
7 is -- Los Angeles is surrounded by mountains,  
8 and so you have literally a basin that is --  
9 you've got the ocean on one side, the  
10:41 10 mountains on the other side, and air sweeps in  
11 and sweeps out, but you've got essentially a  
12 big chemical retort in that basin.

13 Q. Okay.

14 A. You've got sunlight that comes in,  
10:41 15 you've got emissions that are released from  
16 automobiles and a lot of chemical reactions,  
17 atmospheric chemical reactions occur. Okay?  
18 So you've got a pretty good spectrum of  
19 chemicals in the environment there. Some of  
10:41 20 them are oxidizing, some of them are not. But  
21 one of the big questions was, what do we know  
22 about the carcinogenicity of a lot of these  
23 molecules that have been identified.

24 For example, automobiles prior to  
10:41 25 the catalytic converter put out a lot of

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10:41 1 hydrocarbons. What was the carcinogenic  
2 potential of the hydrocarbons, what do we know  
3 about the carcinogenic potential of  
4 hydrocarbons. So this was a survey that we  
10:42 5 made not -- I'm using hydrocarbons as a  
6 specific example, but it was a survey of that  
7 nature.

8 Q. But it was focused generally on  
9 the pollutants that were in that particular  
10:42 10 environment?

11 A. That had been identified in that  
12 particular environment, that's correct.

13 Q. Including hydrocarbons and other,  
14 I guess, emissions from industry and things  
10:42 15 like that?

16 A. Well, it was not only that, but it  
17 was the things that might have been -- might  
18 be secondary pollutants that had been -- as I  
19 said, you have a chemical reaction process  
10:42 20 where you've got hydrocarbons and nitric  
21 oxide. You've got ultraviolet light. You  
22 wind up with nitrogen dioxide, ozone and  
23 various asexual peroxides, all of which are  
24 quite reactive, and the question is, what do  
10:43 25 we know about the carcinogenicity of those

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10:43 1 compounds.

2 Q. In making that evaluation did you  
3 study the potential carcinogenic effects of  
4 the compounds that you started with or the  
10:43 5 compounds that you've just described that  
6 ultimately were in the air?

7 A. I'm not sure I understand that.  
8 The carcinogenicity of -- well, the starting  
9 compound, as I understand the question, the  
10:43 10 starting compound would be gasoline.

11 Q. Okay.

12 A. I mean, this is being -- this is  
13 what goes into the internal combustion  
14 engine. What comes out is hydrocarbons and  
10:43 15 various oxides.

16 Q. So -- and that's exactly what I  
17 was getting at, is to test the health effects  
18 of -- of what comes out, the product of the  
19 combustion in the engine. I assume you didn't  
10:44 20 start by testing the carcinogenicity of --  
21 can't speak today -- of gasoline?

22 A. That has been tested, by the way.

23 Q. Okay.

24 A. What --

10:44 25 Q. But you understand my question?

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1 44 1 A. The -- the -- I understand. What  
2 you -- what was done, other people -- and I  
3 can't tell you the exact organization, but  
4 there have been tests of the carcinogenic  
10:44 5 nature of gasoline vapor, for example.

6 Q. Uh-huh. Was that one of the  
7 components that you investigated in this --

8 A. I don't recall if it actually went  
9 into this report or not. It may have come out  
10:44 10 after this report was done.

11 Q. Okay.

12 A. That's not a -- it was a report  
13 that was done, it seemed to indicate an  
14 increase in kidney cancer, in people who were  
10:44 15 exposed to this, or in animals who were  
16 exposed to that vapor. But I don't recall  
17 those studies, but --

18 Q. But your study focused on what --

19 A. Not only what came out of the  
10:45 20 exhaust, but then what got -- how it got  
21 transformed in the atmosphere, because these  
22 reactions really continue to go on. That's  
23 really the important point. You're dumping  
24 things out of the exhaust of the automobile.

10:45 25 Q. Right. You start with gas, you

51710 1131

10:45 1 burn it?

2 A. That's right.

3 Q. And then something comes out of  
4 the exhaust pipe?

10:45 5 A. That's right.

6 Q. Okay. Then the process continues?

7 A. And then the process continues. •

8 So it's not only what comes out, but what  
9 happens to it with time. I mean, that has

10:45 10 been studied, too. So you looked at the --

11 Q. Uh-huh.

12 A. -- carcinogenic potential of some  
13 of these things, too.

14 Q. And if I understand the goal of  
10:45 15 the work that you did in the publication we  
16 were discussing, which is Number 11 and 19,  
17 your focus was on those end products, those  
18 things that were the ultimate result of that  
19 chain of events, including the changes that

10:46 20 occurred even after they left the tail pipe?

21 A. Our list would have come from --  
22 or our target compounds would have come from  
23 published reports of air pollution in the  
24 South Coast Air Basin.

10:46 25 Q. Uh-huh.

51710 1132

1 A. I was trying to describe the  
2 source of this. Some of these just came from  
3 emissions from plants, too. But, I mean --

4 Q. Sure.

10:46 5 A. -- whatever had been reported in  
6 that -- in the atmosphere.

7 Q. From a toxicological standpoint,  
8 though, and let's focus -- let's move for a  
9 second from the atmosphere to the human body.

10:46 10 The type of process that you just described  
11 also goes on in the human body; is that not  
12 correct? Chemical substances change?

13 A. Right. You ingest nutrients, they  
14 go through biochemical reactions and are  
10:46 15 reduced to energy, right.

16 Q. And there are -- there are  
17 numerous steps along the way?

18 A. Many steps, yes.

19 Q. Okay. And I assume that part of  
10:47 20 the work that you did when you tested drugs or  
21 were involved in the testing of the -- what  
22 was it, the cardiac glycerides?

23 A. Cardiac glycosides.

24 Q. Cardiac glycosides, I'm sorry --  
10:47 25 was looking at not just the drug itself, but

51710 1133

10:47 1 what happened to it in the body and how the  
2 metabolites might affect or have undesired  
3 effects on the patient?

10:47 4 A. The -- that process is -- is  
5 interesting. You punched a button that's  
6 intrigued me. Yeah, you -- you're certainly  
7 interested in the metabolite of a drug. As a  
8 matter of fact, you even patent it. You don't  
9 take a patent out just on a parent compound.  
10:48 10 You patent the metabolites. One of the first  
11 things you do is you stuff the drug in an old  
12 male rat and you collect everything that comes  
13 out and you identify it and you take out a  
14 patent on it.

10:48 15 Q. Now, why is it -- as a layman, why  
16 is it that -- if a company makes something you  
17 put in a rat, why should that company be  
18 entitled to patent what comes out?

10:48 19 A. The -- well, can I give you an  
20 example?

21 Q. Absolutely.

22 A. Borroughs Wellcome -- they are no  
23 longer Borroughs Wellcome, unfortunately.  
24 Borroughs Wellcome made a drug called -- they  
10:48 25 marketed as Zylprim. It was at that time the

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1 only drug in the world that could treat gout,  
2 that was effective against gout.

3 Q. Uh-huh.

4 A. The chemical name or the generic  
10:49 5 name would be allopurinol. Now, a very funny  
6 thing happened. When you put allopurinol in a  
7 rat, you couldn't find it. All you find was  
8 oxypurinol, the metabolite. Well, it turned  
9 out that it was the oxypurinol that was active  
10:49 10 against the gout, not the allopurinol. In the  
11 absence of a patent on oxypurinol, Borroughs  
12 Wellcome would have had allopurinol. Their  
13 competitor could have gone after oxypurinol.

14 Q. Uh-huh.

10:49 15 A. Therefore, you are allowed to  
16 patent the metabolites, not knowing what the  
17 active form of that metabolite -- of that drug  
18 might be.

19 Q. And is that because the -- you  
10:50 20 know, those chemicals that are in the chain  
21 downstream from a drug that you administer are  
22 there because of the drug that you administer?

23 A. I don't understand that question.  
24 I'm sorry. Try that again.

10:50 25 Q. Well, let me use my -- let me use

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10:50 1 an example.

2 A. All right.

3 Q. If the -- the second chemical that  
4 you described, the -- what was the second one  
10:50 5 that they found?

6 A. Oxypurinol.

7 Q. Oxypurinol. If that was a  
8 substance that was just naturally occurring in  
9 the rat, then any rat that you pulled off the  
10:50 10 shelf had oxypurinol, certainly it wouldn't be  
11 your position that the company would be  
12 entitled to patent oxypurinol?

13 A. Might be.

14 Q. Okay. Why?

10:50 15 A. Because you can -- there's several  
16 different types of patents you can take out.  
17 You can take out a -- what is called a use  
18 patent.

19 Q. Okay.

10:51 20 A. Say you -- you patent a compound  
21 that -- and I don't know what the limit is,  
22 17 years or something like that.

23 Q. Yeah.

24 A. It runs out. But you find another  
10:51 25 specific use for that compound. Nobody knew

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1 51 1 about it, but it can be used to treat  
2 something else.

3 Q. Uh-huh.

4 A. And you have that knowledge, and  
10:51 5 you can establish that. You can patent that  
6 compound for that specific use.

7 Q. Okay.

8 A. So there -- now, and once again,  
9 I'm not an expert in patent law, but when I  
10:51 10 was there at Borroughs Wellcome, I took out a  
11 couple of use patents. And that's the only  
12 reason I know anything about it.

13 Q. Okay. Well, the concept that  
14 we've just been kind of touching on that --  
10:51 15 that at least pharmaceutical companies were  
16 aware that there might be a reason to protect  
17 by patent or certainly to identify and examine  
18 the downstream chemicals, the metabolites of  
19 something in a living organism, is that a  
10:52 20 concept that you were aware of during your  
21 years at RJR?

22 A. I'm aware -- I was not aware of it  
23 when I was with the company earlier --

24 Q. Uh-huh.

10:52 25 A. -- in '68.

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10:52 1 Q. Okay.

2 A. But, of course, after that, I went  
3 with Borroughs Wellcome and other companies,  
4 and I came back to R.J. Reynolds in 1984.

10:52 5 And, of course, it's something that I'm aware  
6 of -- that I learned in the course of my  
7 career. Is that the question?

8 Q. Yeah. But is it something that --  
9 I know that at one point when you were first  
10:52 10 at RJR there was -- one of the things you  
11 worked on had to do with pharmaceuticals?

12 A. Exactly.

13 Q. RJR had a pharmaceutical, I guess,  
14 division --

10:53 15 A. No.

16 Q. -- or was thinking about  
17 developing a pharmaceutical division?

18 A. My recollection is that the  
19 company was preparing to buy a pharmaceutical  
10:53 20 operation. And as a result of that, they were  
21 trying to construct an in-house expertise that  
22 would interface with that. That was my  
23 understanding.

24 Q. Okay.

10:53 25 A. And so we were doing some work on

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10:53 1 compounds that had pharmacological --  
2 potentially good pharmacological activity.

3 Q. Okay. In your studies in Southern  
4 California, the title says that the emphasis  
10:53 5 was on the carcino -- say that word for me.

6 A. Carcinogenicity.

7 Q. Thank you. The carcinogenic  
8 potential --

9 A. Uh-huh.

10:53 10 Q. -- of the substances you tested.  
11 How did you go about making that evaluation?

12 A. The -- there are several ways that  
13 you -- you try to look for carcinogenic  
14 potential. None are particularly

10:54 15 satisfactory. But you -- you do what you  
16 can. You look for biological activity that  
17 might give a clue.

18 In vitro type tests, animal  
19 studies, and there is a field that has emerged  
10:54 20 over the last, believe it or not, last  
21 50 years that is -- hopefully one day will  
22 give us really good -- good evidence. And  
23 it's called quantitative structure activity  
24 relationships.

10:55 25 Q. What's that mean?

10:55 1 A. QSAR is a good way to abbreviate  
2 that. It is the ability to study the  
3 potential biological activity of a molecule by  
4 its structural characteristics.

10:55 5 Q. And just -- I mean, how do you --  
6 how do you go about that? What do you -- what  
7 does that mean? .

8 A. The -- if you had a molecule  
9 that -- let's talk about the food -- food --  
10:55 10 potential in foods. A few years ago a man  
11 from Japan named Sugimura, Dr. Sugimura,  
12 reported that there were certain molecules  
13 that occurred in foods at incredibly low  
14 levels that were incredibly potent in certain  
10:56 15 types of biological testing. One of these was  
16 the so-called Aims Mutagenesis Bioassay.

17 Q. Okay.

18 A. These were the most potent  
19 compounds that had ever been tested in that  
10:56 20 particular test. Now, they are complex  
21 heterocyclic molecules and they contain ring  
22 nitrogen. And it's possible to look at those  
23 in relationship of the potency in that test  
24 with respect to the structure of the  
10:56 25 molecule. So I've got a family of molecules.

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10:56 1 Q. Uh-huh.

2 A. They are somewhat related. And  
3 now, by doing a structure activity  
4 relationship, as I can look at ring  
10:57 5 constituents and ring substituents and relate  
6 that to the potency of the molecule as it is  
7 measured by that test.

8 Q. Uh-huh.

9 A. All right. That can be done. And  
10:57 10 one obtains a very interesting relationship  
11 that tells you something about the structural  
12 requirements for activity. You can also  
13 determine what other structure requirements to  
14 eliminate the activity. So that's what I mean  
10:57 15 by quantitative structure activity  
16 relationship studies. Can you look at the  
17 structure of a molecule, make some educated  
18 prediction as to whether it will possess a  
19 certain type biological activity or not.

10:57 20 We are still struggling with that.

21 Q. Uh-huh. Why does biological  
22 activity matter?

23 A. The -- it matters for a number of  
24 reasons. One, you have to remember a  
10:58 25 nutrient, glucose, has biological activity.

51710 1141 =

10:58 1 It can be utilized in the production of  
2 energy.

3 Q. All right.

4 A. But I --

10:58 5 Q. But that wasn't the kind of  
6 biologic activity you were studying, right?

7 MS. FORBES: Jeff, just let  
8 him finish his answers.

9 MR. THOMPSON: I will, I'm  
10:58 10 just trying to --

11 MS. FORBES: I know.

12 A. No -- well, you asked me why is  
13 biological activity important?

14 Q. (By Mr. Thompson) That is  
10:58 15 correct.

16 A. Okay. So I'm trying to give you  
17 an idea. There's a spectrum of reasons that  
18 it's important. You want to know -- it is  
19 unbelievably rare to find a molecule that has  
10:58 20 one type of biological activity alone, all  
21 right?

22 Q. Uh-huh.

23 A. Now, that activity escalates with  
24 dose, or it changes with dose. If I go down  
10:59 25 to a really low dose, I'm going to see one

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10:59 1 level of biological activity. And I might not  
2 see anything else. If I give a higher dose,  
3 now I might see two or three different types  
4 of biological activity. Give a little bit  
10:59 5 higher dose still, and I will see a little  
6 different type of activity.

7 Q. Uh-huh.

8 A. I keep adding. So, now, think  
9 about what I'm saying, that the biological  
10:59 10 activity that I see is related to the dose  
11 that I gave, not just the structural  
12 component.

13 Q. Uh-huh.

14 A. But what level of dose is required  
10:59 15 to move up another quantum of activity.

16 Q. And when you're talking about  
17 activity, if I understand you correctly,  
18 you're not talking about just quantification,  
19 you're talking about different types?

11:00 20 A. Sure.

21 Q. Okay.

22 A. Think about salt. I give -- I put  
23 one grain of salt, one crystal of salt on your  
24 tongue, it's salty. That's a biological  
11:00 25 activity.

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11:00 1 Q. Uh-huh.

2 A. And I take the spectrum, let's  
3 give you a big dose of salt, all of a sudden  
4 you might have renal hypertension. You might  
11:00 5 have -- all sorts of different responses might  
6 come out of that massive dose.

7 Q. Okay.

8 A. So -- so this goes all the way  
9 back to ancient philosophy in toxicology. The  
11:00 10 dose determines the poison. This was -- this  
11 was attributed to a guy name Paracelsus in the  
12 14th century or something like that. His name  
13 was Theophrastus Bombastus von Hohenheim, which  
14 is why they called him Paracelsus. But at any  
11:01 15 rate, that is a -- sort of an adage, if the  
16 dose makes the poison.

17 Q. Okay.

18 A. And so it's important to know a  
19 spectrum of biological activities -- excuse  
11:01 20 me, the biological activity at a spectrum of  
21 doses.

22 Q. Because a substance does, in fact,  
23 do different things or has potential to do  
24 different things in a living organism at  
11:01 25 different doses?

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1 01 1 A. That's correct.

2 Q. What kind of biologic activity did

3 the Aims test?

4 A. The -- the test really tested for

11:01 5 the ability of a chemical to produce a reverse

6 mutation.

7 Q. What does that mean?

8 A. In a -- okay. Dr. Aims, Dr. Bruce

9 Aims at U Cal Berkley had developed -- and now

11:02 10 I don't know how many strings of these things

11 are. He was working with salmonella type of

12 murine bacteria, in which he created an

13 organism that was histidine dependant. That's

14 an amino acid.

11:02 15 Now, this is a mutation. This is

16 a forward mutation. He's created a mutation

17 in the bug. What you're doing is you're

18 testing for the ability of a chemical to send

19 it back to the original form.

11:02 20 Q. Uh-huh.

21 A. If you do not get that, then you

22 don't get a proliferation of the colonies on a

23 plate you are looking at.

24 Q. Uh-huh.

11:02 25 A. So it is a test for the ability of

51710 1145

11:02 1 a chemical to cause a reverse mutation in  
2 salmonella in that particular -- in some --  
3 not a particular string. I must be -- I don't  
4 know how many strings there are now that they  
11:03 5 use for this type of testing.

6 Q. Uh-huh. Why is -- why is it  
7 important that a substance either does or does  
8 not cause mutations in a string of bacteria?

9 A. The original idea -- and I believe  
11:03 10 that this test was published in the  
11 proceedings of the National Academy of  
12 Sciences somewhere around 1974, '73 or '74,  
13 something like that. It was originally  
14 believed that this was going to produce really  
11:03 15 good evidence on the ability of a chemical to  
16 alter the genetic potency -- genetic ability  
17 of a cell.

18 Q. Uh-huh.

19 A. All right. Which would be  
11:03 20 ostensibly a wonderful short-term test for  
21 cancer for the carcinogenic potential of that  
22 molecule. And there were some pretty  
23 extravagant claims made early on for the  
24 test. And a lot of us were really -- I mean,  
11:04 25 I was one of them. I thought, this is going

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04 1 to be terrific.

2 It turned out that it was just not  
3 a good predictor for carcinogens. And  
4 Dr. Aims is, I think, now one of these people  
11:04 5 who just says, you know, it didn't work. It's  
6 a biological activity. It's about a 50/50.  
7 It's probably no better than a coin toss now.

8 Q. Uh-huh.

9 A. And yet it's still a really  
11:04 10 interesting test. I mean, it certainly hadn't  
11 gone away. It's still out there. And I think  
12 someday perhaps we will learn something from  
13 it. I mean, there are a lot of strings still  
14 being developed in this area.

11:05 15 Q. Uh-huh. Was the Aims testing the  
16 primary type of test that you utilized in your  
17 1979 work?

18 A. Can I see that just a minute,  
19 please?

11:05 20 Q. Sure.

21 A. And this is Number 11?

22 Q. Yes, sir.

23 A. Mr. Thompson, I don't recall. I  
24 would have to go back and look at it. I don't  
11:05 25 recall what it is. I -- from the date, 1979,

51710 1147

11:05 1 this is only about four or five years after  
2 the test became -- was published.

3 Q. Uh-huh.

4 A. There weren't many strings  
11:05 5 available, not many people could really do the  
6 test very well, and I doubt there was that  
7 much of that data available. We probably  
8 looked at animal data. I'm just speculating.

9 Q. Okay. Using that test as a model,  
11:06 10 as a hypothetical for a second, if -- we've  
11 talked about metabolites, we've talked about  
12 cars burning gas and chemicals downstream.  
13 One would have to test the substance that is  
14 biologically active in order for that -- for  
11:06 15 any assay to be effective? Do you know what  
16 I'm getting at?

17 THE WITNESS: If you don't --

18 Q. (By Mr. Thompson) For example --

19 A. If you don't have a molecule in  
11:06 20 your mixture that possesses biological  
21 activity, then you're not going to see it in  
22 that particular test.

23 Q. Right.

24 A. Okay.

11:06 25 Q. So if we -- let's use our Southern

51710 1148

1 06 1 California example again. If we used gasoline  
2 molecules in an assay, like the Aims test --

3 A. Uh-huh.

4 Q. -- and it was positive or  
11:07 5 negative, that wouldn't tell us anything about  
6 whether a molecule that was the ultimate  
7 result of combustion, interaction with  
8 constituents of the atmosphere had biologic  
9 activity?

11:07 10 A. I think what you said is correct.  
11 I'm going to try -- I'm going to try to get at  
12 it another way and see if it's correct. If --  
13 okay. If I tested gasoline and I -- and take  
14 a specific test, the test for a specific  
11:07 15 biological activity, and the gasoline didn't  
16 show me anything, it showed no positive  
17 response, that would not tell me whether or  
18 not one of its oxidation products was going to  
19 have biological activity in that particular  
11:08 20 test.

21 Q. Uh-huh.

22 A. It certainly would not -- I would  
23 not reliably predict based on the guy -- on  
24 say that particular hydrocarbon test about its  
11:08 25 products. I would want to test its products,

51710 1149

11:08 1 but maybe not necessarily in that test.

2 Q. Right.

3 A. I mean, I --

4 Q. But the point is that in order to  
11:08 5 make any judgement at all about the products,  
6 ultimately you would want to identify and test  
7 those products?

8 A. Unless I had a really good --  
9 sooner or later I would get the testing. But  
11:09 10 what would guide me to the testing? I would  
11 use some predictive methodology to give me  
12 a -- a hierarchy, that is, a priority list of  
13 what do I test, what do I -- what do I look  
14 at. Remembering that a lot of the products  
11:09 15 that come out of that exhaust are very  
16 unstable molecules. They come apart pretty  
17 quickly (indicating). They are not in the  
18 atmosphere very long and --

19 Q. Uh-huh. It's hard to catch them?

11:09 20 A. Well, it's -- you can catch them,  
21 but you can't hold them. They disintegrate on  
22 you.

23 Q. Okay.

24 A. So it's sort of difficult to test  
11:09 25 some of them. But believe it or not, tests

51710 1150

1 09 1 have been done on them.

2 Q. You previously referred --

3 MR. THOMPSON: Well, we need  
4 to go off the record. I'm sorry.

11:10 5 THE VIDEOGRAPHER: Off the  
6 video record.

7 (A recess was taken.)

8 THE VIDEOGRAPHER: We're on  
9 the video record.

11:19 10 Q. (By Mr. Thompson) Doctor, we're  
11 back after a break. I just want to follow up  
12 a minute on our kind of detailed discussion of  
13 chemicals and their products. You mentioned  
14 briefly a new area of studies regarding  
11:19 15 chemicals and cancer that dealt with the  
16 shape --

17 A. Structure.

18 Q. -- structure of the molecule,  
19 correct?

11:20 20 A. Right.

21 Q. And would you agree with me that  
22 the same thing is true of that type of assay  
23 or that type of analysis that -- that in  
24 determining whether or not something could  
11:20 25 cause cancer, using that assay, that you need

51710 1151

11:20 1 to test or examine the shape of the substance  
2 that is the product that you're -- that is of  
3 concern? Let me use a concrete example  
4 because that will help us. Benzene.

11:20 5 A. Okay.

6 Q. Do you agree, in your opinion, is  
7 benzene a human carcinogen?

8 A. It has been characterized as a  
9 human carcinogen. It believe it has thrown  
11:21 10 some -- it has shown some blood dyscrasias in  
11 animals, in animal studies, so -- and  
12 epidemiologically has been shown to be  
13 associated with leukemia, yeah.

14 Q. Okay. Are you familiar at all  
11:21 15 with the investigation as to the actual  
16 molecule -- the molecular issue with regard to  
17 benzene and its carcinogenic potential?

18 A. I haven't studied specifically  
19 benzene.

11:21 20 Q. Right.

21 A. But I know that the curiosity goes  
22 like this. Benzene is a very unremarkable  
23 molecule, very simple molecule.

24 Q. Just a ring?

11:21 25 A. But it is said to be an aromatic

51710 1152

11:22 1 molecule, meaning that it has six delocalized  
2 electrons in that range.

3 Q. And why does that matter?

4 A. This gives it a reactivity that  
11:22 5 you would not find in a molecule that had  
6 exactly the same number of carbons in a ring  
7 structure, in other words, a six-carbon ring.  
8 that did not have delocalized electrons, did  
9 not have resonating electrons, resonate  
11:22 10 structures.

11 Q. Uh-huh.

12 A. We would call such a molecule a  
13 saturated molecule. In other words, all of  
14 the available bonding electrons had hydrogens  
11:22 15 on it.

16 Q. Uh-huh.

17 A. Now, that is a more stable  
18 molecule, and it has a little bit different  
19 shape. Benzene is a planer molecule. In  
11:22 20 other words, benzene ring would lie flat --  
21 flat on the table.

22 Q. Uh-huh.

23 A. A similar structure, a six-carbon,  
24 would have what we call a chair form. It  
11:23 25 would not lie flat in the plane.

51710 1153

11:23 1 Q. Uh-huh.

2 A. Now, our understanding of  
3 metabolism is leading us to understand that  
4 the shapes play a role in biological  
11:23 5 activity. So just to say that it's an  
6 unremarkable simple molecule is really not  
7 sufficient. There's a certain aromaticity to  
8 that. It's said to be a resonating  
9 structure.

11:23 10 Q. And isn't it correct, or am I  
11 correct in the statement that the reason that  
12 that is important is because the reactivity of  
13 the molecule is at least a factor that can  
14 lead to the formation of other types of  
11:24 15 molecules as a product of reaction with  
16 benzene, for example?

17 A. I have -- once again, you're  
18 leading me into an area where I'm -- I'm not  
19 up to date. I mean, I haven't gone back and  
11:24 20 reviewed that. But benzene can be  
21 metabolized, as I recall, to phenol, and I'm  
22 not sure what other -- I'm not sure what the  
23 ring opening metabolite is like. I don't know  
24 if it opens or not. I don't recall that.

11:24 25 Q. Uh-huh. Are you familiar at all

51710 1154

11:24 1 with research or studies relating to the shape  
2 or structure of certain benzene metabolites  
3 and their cancer-causing ability?

4 A. Can you be specific? Can you  
11:24 5 tell -- can you give me an idea of what  
6 metabolites you're talking about?

7 Q. Well, I just thought that -- you  
8 had mentioned this specific area of science.

9 A. Uh-huh.

11:25 10 Q. And it, in fact, turns out that it  
11 is -- that it is metabolites of benzene, and  
12 in some cases, distant metabolites of benzene  
13 that appear to have the shape or the  
14 structures that you, I think, were alluding to  
11:25 15 earlier, molecules that have bays, molecules  
16 that have open --

17 A. Okay.

18 Q. -- radicals --

19 A. Okay. I know where you're going.

20 Q. Okay.

21 A. I understand what you're -- what  
22 you're saying now. You're referring to the  
23 polycyclic molecules, which are --

24 Q. Uh-huh.

11:25 25 A. Polycyclics are not metabolites of

51710 1155

11:25 1 benzene. The body does not convert benzene to  
2 a polycyclic.

3 Q. Okay.

4 A. Okay. There was a point that I  
11:25 5 wanted to make just then that you had --

6 Q. Are you aware of any study,  
7 research, that specifically has identified the  
8 mechanism by which benzene causes cancer?

9 A. I am not familiar with -- I don't  
11:26 10 know of, and I've looked for, any study which  
11 shows a mechanism by which any molecule causes  
12 cancer. I just don't know that that exists.  
13 I have looked for it. It's one of the things  
14 that we would most like to have is that  
11:26 15 mechanism.

16 Q. Okay. In presenting your opinions  
17 today, your testimony, are the opinions you're  
18 presenting your own opinions about smoking and  
19 health?

11:27 20 A. They're certainly my opinions,  
21 yes, sir.

22 Q. Okay. As opposed to your  
23 company's opinions?

24 A. I don't know that my company has  
11:27 25 opinions that are different from mine.

51710 1156

11:27 1 Q. Okay.

2 A. I mean, I --

3 Q. I --

4 A. Our opinions are consistent,  
11:27 5 mutually consistent.

6 Q. Okay. And that's what I -- you  
7 know, is there any specific issue on which  
8 you, William Simmons, expert, disagrees with  
9 the -- the official position of RJR in the  
11:27 10 area of smoking and health?

11 A. Not that I know of.

12 Q. For example, RJR's position is  
13 smoking does not cause lung cancer; is that  
14 correct?

11:27 15 A. No, sir.

16 Q. That's not correct?

17 A. No, sir.

18 Q. What is their position?

19 A. The position, for as long as I've  
11:28 20 been aware, is that smoking may cause lung  
21 cancer. It's possible that it does. But it  
22 has not been scientifically proven that it --  
23 that it does. You have on one hand  
24 epidemiological evidence which is extensive  
11:28 25 and around the world. On the other hand, you

51710 1157

11:28 1 have a lot of animal studies which have been  
2 done by inhalation of cigarette smoke which  
3 have not demonstrated that cigarette smoke  
4 causes lung cancer.

11:28 5 Now, this lack of convergence of  
6 these two major lines of evidence is the basis  
7 for the position that while it -- smoking may  
8 cause these diseases, cause the disease of  
9 lung cancer, it has not been proved to a  
11:29 10 scientific certainty that it does cause lung  
11 cancer.

12 Q. Okay.

13 A. So that's different from making  
14 the statement that you made that it does not  
11:29 15 cause lung cancer.

16 Q. Okay. And that's not RJR's  
17 position?

18 A. And has not been for as long as  
19 I've been aware.

11:29 20 Q. Okay. And it's not your position,  
21 either?

22 A. That's correct.

23 Q. Do you believe smoking causes lung  
24 cancer?

11:29 25 A. I do not know whether it does or

51710 1158

1 29 1 not. I don't know whether it will ultimately  
2 be shown that it does cause lung cancer. I  
3 don't know whether it will ultimately be shown  
4 that it is a factor coupled with other factors  
11:29 5 that causes lung cancer.

6 The evidence with respect to lung  
7 cancer and risk factors is becoming broader,  
8 more diffuse, and not more concentrated. In  
9 other words, we -- we know of a lot of risk  
11:30 10 factors for lung cancer now. And that has  
11 evolved over a period of a number of years.  
12 So I don't know where it's going to come out.  
13 I don't know whether it is a specific cause or  
14 not.

11:30 15 Q. If I asked you in your opinion  
16 whether it was more likely than not that  
17 smoking was a cause of lung cancer in humans,  
18 what would your answer be?

19 A. I don't know how to partition the  
11:30 20 probability.

21 Q. So --

22 A. I don't have a way of partitioning  
23 the probability.

24 Q. So to you the question -- the only  
11:31 25 question that needs to be answered is, does it

51710 1159

11:31 1 cause cancer to a level of -- can it be  
2 established that it causes cancer at a level  
3 of scientific certainty?

4 A. It has to meet -- your evidence --  
11:31 5 think of -- think about what the process is  
6 like at this point. We have had  
7 epidemiological evidence since 1950. The  
8 first associations were published both  
9 Dr. Dahl and Dr. Hertz Wynder published in  
11:31 10 1950 this association. And then later in the  
11 '50s, skin painting studies were done by  
12 Dr. Wynder showing that tar painted on the  
13 skin of animals produced tumors.

14 But this was not satisfactory,  
11:32 15 this '64 surgeon general's report, they even  
16 call for inhalation studies. Let's get good  
17 inhalation data.

18 Now, attempts have been made for a  
19 number of years by a number of different  
11:32 20 organizations around the world going from  
21 Betel, Geneva to Albuquerque, New Mexico to  
22 produce lung tumors in animals with cigarette  
23 smoke. They have not succeeded.

24 However, if you look at other  
11:32 25 chemicals that have been tested in animals,

51710 1160

1 bischloromethylether, vinyl chloride, and now  
2 diesel exhaust, you find that these chemicals  
3 or these mixtures of chemicals will produce  
4 lung tumors. Why?

11:33 5 We don't have a mechanism. I  
6 don't -- I don't have a mechanism even for  
7 chemicals that produce lung cancer. I don't  
8 know what the mechanism is by which it  
9 produces that lung cancer. But because I have  
11:33 10 converging information, that is, I have  
11 epidemiology in humans, and I have inhalation  
12 exposures in animals, both producing evidence  
13 of lung carcinogenicity, then I can conclude  
14 that these things are lung carcinogens.

11:33 15 Now, go back to cigarette smoke.  
16 We have a long record of animal studies which  
17 have failed to produce evidence. So now  
18 you're faced with another problem. Not only  
19 have you got to do an animal study -- would I  
11:34 20 like to see an animal study in which I've got  
21 an excess of lung cancer, I want an  
22 explanation as to why the previous studies  
23 didn't produce it. I mean, a built-up -- a  
24 confusing body of evidence -- I probably am  
11:34 25 not doing a good job explaining that. But

51710 1161

11:34 1 that's -- therein lies the problem. What is  
2 the problem? I don't know.

3 Q. But your search is for a level of  
4 proof that allows you to say with certainty  
11:34 5 that it does, in fact, cause lung cancer?

6 A. I would like to see -- yes, I  
7 would like to see evidence that would allow me  
8 to say, yes, I can now conclude that it  
9 produces -- it causes lung cancer in humans.

11:35 10 Q. And in your opinion, any quantum  
11 of proof less than that is not something that  
12 is appropriate for making public health  
13 decisions?

14 A. I don't make public health  
11:35 15 decisions. I'm going from a scientific  
16 standpoint.

17 Q. Okay.

18 A. Public health scientists may have  
19 a different level of requirement for making a  
11:35 20 public health pronouncement.

21 Q. And why is that?

22 A. The public health scientist is --  
23 a public health official will make his  
24 pronouncement based on his judgment as to what  
11:35 25 might be best for the population. And so he

51710 1162

11:35 1 can make that pronouncement. But it's not  
2 necessarily a scientific pronouncement. It is  
3 a matter of his judgment.

11:36 4 Q. What should a manufacturer of  
5 products use as a quantum of proof, in your  
6 opinion?

11:36 7 A. I can only speak as a scientist.  
8 what my views as a scientist are. I work for  
9 a manufacturer, but I render an opinion based  
10 on scientific evidence and a scientific  
11 conclusion. So I -- I don't know how to  
12 answer your question. I mean, all I can do is  
13 tell you what my opinion is, what my judgment  
14 is, based on the science.

11:36 15 Q. But isn't your job to tell your  
16 company what your opinion is based on the  
17 science?

18 A. And I do that, yes, that's  
19 correct.

11:37 20 Q. And what is it that -- that you,  
21 in your opinion -- I mean, what is it that you  
22 believe they should do with that information?

23 A. I don't -- I don't make management  
24 decisions. I mean, my job is to tell them  
11:37 25 what I think, what my conclusions are. And I

51710 1163

11:37 1 don't try to tell them what to do with it. I  
2 mean, I-just give them the information.

3 Q. And so earlier you said that  
4 smoking may cause lung cancer?

11:37 5 A. It might.

6 Q. In your opinion, what should your  
7 employer do with that information? How should  
8 they act based on that information?

9 A. You're asking me to make a  
11:37 10 management decision. I can tell you what they  
11 have done. I mean, is that a fair thing to  
12 do?

13 Q. Sure.

14 A. If you look at the -- at the  
11:38 15 way -- I can't speak for the whole industry,  
16 but the way my company has responded for many  
17 years over that issue, they have evolved  
18 cigarette design in such a way that both tar  
19 and nicotine have been reduced rather  
11:38 20 dramatically from, say, 1950 to the present.  
21 There's a so-called sales weighted average  
22 weight of tar and nicotine in our products has  
23 come down dramatically. Well, why is this? I  
24 mean, what is the point here?

11:38 25 Well, the idea is, as I pointed

51710 1164

11:38 1 out before, the dose makes the poison. If, in  
2 fact, it turns out that cigarettes do produce  
3 lung -- do generate lung cancer in smokers,  
4 then the idea would be to reduce the dose as  
11:39 5 far as you can, reduce the -- whatever the  
6 yield is on the cigarettes.

7 Not being given a specific target  
8 by any member of the scientific community, the  
9 target has shifted from polycyclic aromatic  
11:39 10 hydrocarbons to phenols to all -- all sorts of  
11 compounds have been listed as potential agents  
12 that might cause cancer in smokers. We have  
13 reduced the yield of these compounds across  
14 the board. Everything has come down over the  
11:39 15 past 45, 46 years.

16 Now, knowing that we had done  
17 that, we had looked at design, once again, I'm  
18 not the expert in this area. Dr. Dave  
19 Townsend is the guy who's really expert in  
11:40 20 that design and knows the evolution better  
21 than I do. But the one thing that I do know  
22 is, we had -- the company, I think, had  
23 reached the position where we had hit a limit  
24 as far as to tobacco-burning cigarettes go.

11:40 25 You burn cigarettes, you produce

51710 1165

11:40 1 products of incomplete combustion and the idea  
2 was, okay, well, let's just produce a  
3 cigarette that doesn't burn tobacco at all.

4 Q. Premier?

11:40 5 A. Premier. And so we tried with  
6 Premier. Hard. It was not acceptable to the  
7 smoker. Smokers didn't find it acceptable. .

8 Q. When you say they didn't find it  
9 acceptable, they -- they wouldn't buy the  
11:40 10 product?

11 A. We couldn't establish a place in  
12 the market. That's --

13 Q. Okay.

14 A. -- essentially what I'm saying is  
11:41 15 that you just -- you just couldn't -- it had  
16 some -- it's different. It had some  
17 differences --

18 Q. Uh-huh.

19 A. -- that apparently the smoker  
11:41 20 could not adjust to. But that doesn't mean  
21 that we've stopped that process. We're  
22 going -- we're trying again now with the -- a  
23 product called Eclipse.

24 Q. Uh-huh.

11:41 25 A. And so this is the way, at least

51710 1166

11:41 1 my company has addressed that possibility over  
2 the years; that is, to reduce the dose, that  
3 is, the quantity of products of incomplete  
4 combustion and then try to go to a cigarette  
11:41 5 that just simply doesn't burn tobacco at all.

6 Q. But if the company is concerned  
7 enough to make those kinds of efforts, why is  
8 not the responsible thing to do to stop  
9 selling the suspect product until you get the  
11:42 10 answer that you're looking for?

11 A. I don't know that that's a  
12 responsible thing to do. I mean, people enjoy  
13 smoking cigarettes. They are apparently well  
14 informed about the hazards of cigarettes; that  
11:42 15 is, the risks that have been published as  
16 being associated with smoking cigarettes. I  
17 don't know that -- I mean, I don't understand  
18 the question. I guess I don't understand the  
19 question. I don't know that that would be the  
11:42 20 responsible thing to do.

21 Q. Okay. In your opinion, it  
22 wouldn't be the responsible thing to do, to  
23 stop selling a product that might cause cancer  
24 until you know?

11:42 25 A. I don't even know how to address

51710 1167

11:43 1 that. I mean -- I don't know. I just don't  
2 know how to answer the question. I'm sorry.

3 Q. Well, I guess if you would, answer  
4 it as a -- as somebody who's been in the  
11:43 5 industry 13-plus years. I mean, how do you  
6 feel personally about selling a product that  
7 might cause lung cancer, in your own opinion?

8 A. In my opinion, I know the  
9 evidence. I know the associated risks. I  
11:43 10 smoke. I smoke between one to two packs a  
11 day. And I understand those risks. I  
12 understand the evidence. I don't know that  
13 that answers your questions, but --

14 Q. So it isn't something that you  
11:44 15 have given thought to that you are involved in  
16 marketing of a product that in your own  
17 opinion may kill people?

18 A. Cigarettes are a legal product.  
19 They are part of a culture. Tobacco use is  
11:44 20 part of the culture of this company for --  
21 from its inception.

22 An example, my earliest ancestor  
23 in this country came to Virginia in 1635 and  
24 grew tobacco. King James I of England issued  
11:44 25 a pamphlet or a -- in 1620 or thereabouts

51710 1168

11:44 1 against tobacco. The health hazards  
2 associated -- the health risks associated with  
3 the use of tobacco in virtually all forms has  
4 been known for an awfully long time, been  
11:45 5 highly publicized. And yet, cigarettes are a  
6 legal product. Millions of people enjoy  
7 smoking. I'm one of them.

8 Is that irresponsible?

9 Q. Is it?

11:45 10 A. Not for me. It may be for you or  
11 for others, but not for me. It's my choice.

12 Q. So in your opinion, your employer  
13 ought to continue selling products, cigarette  
14 products, as long as they're legal, regardless  
11:45 15 of what the scientific literature or data that  
16 you review reveals?

17 A. The -- I think the responsible  
18 thing is to continue to evaluate the evidence,  
19 to study the evidence, and continue to evolve  
11:46 20 the product to make better products, to try to  
21 analyze ways of producing products that may  
22 not have those risks associated with them. I  
23 think that's the responsible thing for a  
24 company to do. And that, in fact, is what my  
11:46 25 company has done.

51710 1169

11:46 1 Q. Tried to build a safer cigarette?

2 A. Tried to build a cigarette that  
3 reduced the yield of products of incomplete  
4 combustion, assuming that the risks associated  
11:46 5 with smoking actually devolve from those  
6 products of incomplete combustion.

7 Now, the scientific community  
8 seems to believe that. There seems to be a  
9 consensus that the health risk may come from  
11:46 10 the tar. I mean, this has been something that  
11 I have seen rather consistently in the  
12 literature.

13 Q. Uh-huh. But kind of looking back  
14 to our previous discussion, tar is not -- you  
11:47 15 know, there are not -- it's not a homogenous,  
16 you know -- there isn't a molecule called tar,  
17 isn't there?

18 A. No, there's not.

19 Q. It's a mixture of things?

11:47 20 A. It's a mixture generally of  
21 products of incomplete combustion of tobacco,  
22 you know. And all of the components that have  
23 been -- or the constituents which have been  
24 reported in tobacco smoke or in cigarette  
11:47 25 smoke have not been identified, say, in every

51710 1170

11:47 1 cigarette. I mean, you -- this list has grown  
2 over a period of 40 years, I guess, of things  
3 that have been identified in -- in cigarette  
4 smoke.

11:47 5 Q. Uh-huh.

6 A. And so things that were reported  
7 in cigarette smoke 40 years ago in very small  
8 levels may not be there today, and vice  
9 versa. There may have been things not  
11:48 10 reported that are there today. But, I mean,  
11 we don't -- what we do know is that the  
12 overall yield has come down over the years  
13 from 33 milligrams of tar, thereabouts, down  
14 to a sales weighted average of about 11 today.

11:48 15 Q. And when you talk about yield,  
16 you're talking really about two things, tar  
17 and nicotine?

18 A. That is correct.

19 Q. Tested under the FTC method?

11:48 20 A. That's correct, yes.

21 Q. Would you agree with me, though,  
22 that to get the answer to your question, the  
23 question that you've posed, which is how --  
24 the mechanism question.

11:48 25 A. Okay.

51710 1171

11:48 1 Q. And the second question that you  
2 believe is unanswered, which is to make a --  
3 re-create the cancer formation, the process of  
4 cancer in an animal model, that one would have  
11:49 5 to first identify the specific constituents of  
6 smoke that are -- that are the causes of  
7 cancer.

8 A. Think about diesel exhaust. If  
9 you expose animals, laboratory animals,  
11:49 10 rodents in this case, to diesel exhaust for,  
11 say, a chronic study, a two-year study in  
12 these animals, what you find is that you have  
13 an excess of lung tumors in animals exposed to  
14 diesel exhaust as opposed to animals that were  
11:49 15 exposed to -- had been exposed to fresh air.

16 Now, what in the diesel exhaust is  
17 responsible for this? I now have an excess of  
18 lung cancer, but I don't have a mechanism by  
19 which the diesel exhaust caused it, nor do I  
11:49 20 know which constituent or constituents of  
21 diesel exhaust are responsible. And yet I can  
22 say that diesel exhaust, as a class of -- a  
23 mixture of compounds, at least in that  
24 experiment at that dose is a carcinogen.

11:50 25 Q. Okay. We talked about your role

51710 1172

11:50 1 as company spokesman. And I think you told me  
2 that it wasn't your understanding that that  
3 was your role. But at the same time, over the  
4 years you have responded publicly in the  
11:50 5 press, in the print media, to various, I  
6 guess, studies that were published or other  
7 public statements --

8 A. That's correct.

9 Q. -- on issues of smoking and  
11:50 10 health.

11 A. Let me just think about that a  
12 minute. I have -- I did some public  
13 appearances when Premier came out that were in  
14 line with the -- with the Premier launch --

11:51 15 Q. Uh-huh.

16 A. -- and so forth. Is that what  
17 you're referring to?

18 Q. That's -- that's part of it. In  
19 making -- I don't know if "press conference"  
11:51 20 is the right word, but there were public  
21 statements made about the nature of the  
22 product and the -- the work that was being  
23 done, and you were part of that?

24 A. And I was a part of that, that's  
11:51 25 correct.

51710 1173

11:51 1 Q. And you were part of that in your  
2 capacity as -- then I think your title was  
3 director of science and health?

4 A. No, smoking and health.

11:51 5 Q. I'm sorry. Smoking and health?

6 A. Actually, actually that's -- let  
7 me go back. Remember I said I took the job in  
8 April of 1986. Remember that?

9 Q. I do remember that.

11:51 10 A. Prior to that, the prior 15  
11 months, I had spent specifically on the  
12 Premier project. Do you follow what I'm --

13 Q. I understand.

14 A. Okay.

11:52 15 Q. Uh-huh.

16 A. And I had done -- I had studied  
17 the scientific literature with regard to  
18 smoking-related health effects. Spent a lot  
19 of time trying to help design Premier so that  
11:52 20 we could reduce or virtually eliminate  
21 products of incomplete combustion of tobacco.  
22 So that was not to say we're not going to use  
23 tobacco in the product. It had plenty of  
24 tobacco in it. We didn't want to burn it. We  
11:52 25 didn't want any products of incomplete

51710 1174

:52 1 combustion.

2 Q. Uh-huh.

3 A. So I knew that product very well.

4 And I knew a great deal about the smoking and

11:52 5 health issue. And consequently, I was chosen

6 to make some statements about Premier in

7 various different types of forums.

8 Q. As an expert on the product?

9 A. As an expert on the product, yes.

11:53 10 Q. In that capacity, though, were

11 you -- I mean, were you just asked to describe

12 your opinions about the product and how it was

13 developed and what the goals were, or were

14 you -- did you work with management to

11:53 15 formulate, in effect, a presentation about the

16 product?

17 A. There were a number of people

18 involved in trying to prepare presentations

19 about the product. I wouldn't necessarily

11:53 20 call them management. The ones I recall were

21 scientists who were involved in that

22 activity.

23 Q. Uh-huh.

24 A. People who had both worked with

11:54 25 the product and tested the product, put the

51710 1175

11:54 1 product through a number of different types of  
2 biological activity testing. So there were a  
3 number of us that worked on those  
4 presentations. And I'm not sure -- you said  
11:54 5 management. I'm not sure what you meant by  
6 "management." These were just my colleagues,  
7 scientific colleagues.

8 Q. Was there any participation by the  
9 public relations department, for example?

11:54 10 A. The -- their participation was in  
11 trying to help the scientists tell the story.  
12 The scientist has a story. He's done some --  
13 development scientist has created a product  
14 and the other scientists have put it through  
11:54 15 different types of tests --

16 Q. Uh-huh.

17 A. -- and so forth. Now, you have a  
18 product that you want to put on the market.

19 Q. Uh-huh.

11:54 20 A. So -- and I'm talking very general  
21 terms, because I don't remember any specifics  
22 here. The public relations people might have  
23 helped us in formulating a way of  
24 communicating to the public. Does that --

11:55 25 Q. Uh-huh.

51710 1176

55 1 A. Does that answer your question?

2 Q. Sure. Help you formulate the  
3 story?

4 A. A message, yes.

11:55 5 Q. In addition to that type of role,  
6 more recently you -- you actually would  
7 respond in the press to the publication of  
8 certain articles, and I've got some -- just  
9 some specifics, and let me see if we can -- in  
11:55 10 1991 there was an article about secondhand  
11 smoke that was probably a news service article  
12 because it was published in several different  
13 places. And you wrote a series of letters to  
14 the editor?

11:55 15 A. Exactly.

16 Q. To the Orlando Sentinel Tribune,  
17 to the Chicago Tribune, to the Washington Post  
18 in which you stated that you disagreed with  
19 that, what was stated in the article?

11:56 20 A. That is correct, yes.

21 Q. And in each case, you were -- you  
22 identified yourself as director of smoking and  
23 health at RJR?

24 A. If it was 1991, I believe that's  
11:56 25 what my title was.

51710 1177

11:56 1 Q. Okay. And so in responding to  
2 these studies, or to these articles, I guess,  
3 discussing the JAMA study, were you expressing  
4 your opinion as a scientist, your personal  
11:56 5 opinion, William Simmons, or were you being  
6 asked to respond on behalf of RJR?

7 A. When I put my name on something,  
8 it's my personal opinion.

9 Q. Okay.

11:56 10 A. Okay? Now, there was a time when  
11 I believed that it might be worthwhile to  
12 write letters to the editor. There was a time  
13 when I believed that -- well, let me give you  
14 an idea what I'm talking about.

11:57 15 The entire environmental tobacco  
16 smoke issue is one that is, in my opinion,  
17 dramatically misreported, not only by the  
18 scientists who work on it, but by the fourth  
19 estate who publishes and comments on it.

11:57 20 And I believed at one time that it  
21 would be worthwhile to prepare a compendium of  
22 scientific articles that had been published,  
23 take them to editorial boards of newspapers,  
24 and inform them, try to educate them as to  
11:58 25 what the science really says, what are the

51710 1178

:58 1 facts here, so that at least when they wrote  
2 editors or wrote comments on environmental  
3 tobacco smoke, they would have the scientific  
4 evidence in their hands.

11:58 5 Q. Like a briefing book?

6 A. It wasn't very brief.

7 Q. A thick briefing book?

8 A. But it was a very big briefing  
9 book, right. I think that may have been a  
11:58 10 failing. We tried to put too much into it. I  
11 think I tried to cover too much ground.

12 Q. Uh-huh.

13 A. But that was a thought that I had  
14 when I was idealistic --

15 Q. Uh-huh.

16 A. -- inform people right, inform  
17 things. Well, so we tried that for a while.  
18 And I did that, yes, I did. I wrote some  
19 letters to the editors and I visited editorial  
11:58 20 boards and so forth on the issue of  
21 environmental tobacco smoke.

22 Q. But those are two different  
23 things. It seems to me that sending  
24 information to an editorial board or providing  
11:59 25 data to somebody is one thing. You've

51710 1179

11:59 1 described that. And I think you've said that  
2 that didn't work, in your opinion?

3 A. Well, I didn't see any positive  
4 results. I didn't see a more balanced article  
11:59 5 based on the science. I didn't see any of  
6 that occurring.

7 Q. Okay. And the second thing there  
8 is -- is, I think, another step, which is  
9 sending a response to be published in the  
11:59 10 paper or magazine or whatever to be read by  
11 the public. I mean, that is -- that becomes  
12 a -- rather than trying to get somebody to  
13 make an objective decision about publication  
14 of things, it becomes using the paper to  
11:59 15 publish your views, a response.

16 A. Okay. So you're referring to  
17 letters to newspapers?

18 Q. Yeah.

19 A. Okay. In scientific circles, in  
12:00 20 scientific areas, it is very common for a  
21 scientist to write a letter to a journal over  
22 a scientific article. Now, as far as I was  
23 concerned, when the newspaper published  
24 something about a scientific article, they had  
12:00 25 stepped into the scientific arena.

51710 1180

11:00

1 Q. Uh-huh.

2 A. And it was my job to write to them  
3 and point out anything I saw that I believed  
4 was scientifically incorrect in that article  
12:00 5 or scientifically deficient.

6 Now, translate it any way you want  
7 to. I mean, that's -- that was my view. This  
8 is a scientific issue. If you are going to  
9 step into science with your newspaper, then  
12:00 10 let's be scientific.

11 Q. And so you've -- you sent  
12 responses and asked them to publish the  
13 responses?

14 A. In some cases, yes, that's right.

12:01

15 Q. At -- is that something you still  
16 do?

17 A. I haven't -- I haven't done that  
18 for about -- I've forgotten the last time I  
19 did it. I don't know how long ago it's been.  
12:01 20 I do not do it anymore.

21 Q. Why not?

22 A. I guess I just got weary,  
23 Mr. Thompson.

24 Q. Nobody was listening?

12:01

25 A. I'm afraid so.

51710 1181

12:01 1 Q. In 1993 you were interviewed on  
2 TV -- is it 1993, Prime Time Live?

3 A. That -- was that Mr. Donaldson?

4 Q. Indeed.

12:01 5 A. Oh, yes, I was.

6 Q. Tell me how that came about.

7 A. That would have been -- I'll take  
8 your word that it was 1993. I don't remember  
9 the date.

12:02 10 Q. I think it was February 25th,  
11 1993.

12 A. It was -- it was February, I'm  
13 pretty sure. Now, it may have been 1993.

14 Q. Okay. How did they come to talk  
12:02 15 to you?

16 A. I'm going to try to get it exactly  
17 right. It was about the time that the  
18 trial -- the Keuper trial was going on in  
19 Belleville, Illinois. And apparently

12:02 20 Dr. Donaldson had indicated to someone, I  
21 don't -- I don't know a lot of details. I  
22 don't understand. But that he was going to do  
23 a so-called expose or something on fraud in  
24 the cigarette industry. I believe, as a  
12:03 25 matter of fact, those are his words. When I

51710 1182

12:03 1 finally met Mr. Donaldson, I think he told me  
2 that. -He asked me, do you know why I'm here.  
3 And I said, I'm not sure. And he said, well,  
4 I'm going to do this program indicating that  
12:03 5 there's fraud in the cigarette industry or  
6 something like that, and has been going on for  
7 a long time. So those were his words. •

8 It involved work that ostensibly  
9 had been done at Reynolds, R.J. Reynolds, at  
12:03 10 least part of this program. Now, I don't know  
11 if this was a Reynolds-specific program. I  
12 didn't even watch the program. But it  
13 involved, for my part, work that had gone on  
14 at R.J. Reynolds between, say, 1968 and 1970.  
12:04 15 And -- okay, you were asking how that came  
16 about? Is that --

17 Q. Yeah, I guess my question -- my  
18 initial question is, why is it that Sam  
19 Donaldson ended up being directed to you?

12:04 20 A. Well, I was -- I guess it was  
21 because I was here and I was at Reynolds in  
22 1968 through 1970. I suppose that's --

23 Q. Okay.

24 A. I guess he wanted to talk to me  
12:04 25 about it.

51710 1183

12:04 1 Q. Okay. And you weren't asked --  
2 your company, your employer didn't ask you to  
3 be the contact person to address  
4 Mr. Donaldson's questions about smoking and  
12:04 5 health generally?

6 A. Not about smoking and health  
7 generally. It was -- I was -- I was the only  
8 person that -- that was really available that  
9 had -- at the scientific level that had been  
12:04 10 working -- that was with the company that had  
11 been with the company at that period of time  
12 that we're talking about.

13 Q. And so it was your understanding  
14 he came to you only because you were somebody  
12:05 15 who had specific knowledge of the events that  
16 occurred in '68, '69, and '70?

17 A. It's my belief that that's the  
18 reason I had to deal with the issue. I was  
19 the only person available who was with the  
12:05 20 company in 1993 that had been with the company  
21 during that period of time that he was  
22 referring to.

23 Q. Okay.

24 A. Now, that's -- to my recollection,  
12:05 25 that's the answer. It's been a while back.

51710 1184

12:05 1 Q. But at any rate, you had the  
2 approval, the -- your employer authorized you  
3 to talk to Mr. Donaldson?

4 A. They certainly -- yes, that's  
12:06 5 correct, yes.

6 Q. Okay.

7 MR. THOMPSON: This is  
8 probably a good place to take a break.

9 THE WITNESS: Boy, it sure  
12:06 10 is.

11 THE VIDEOGRAPHER: Off the  
12 video record.

13 (A recess was taken.)

14 THE VIDEOGRAPHER: We're on  
13:30 15 the video record.

16 Q. (By Mr. Thompson) Doctor, we're  
17 back from lunch. The question I meant to ask  
18 you earlier, just for my information -- we're  
19 in Winston-Salem, North Carolina; is that  
20 correct?

21 A. That's correct, yes, sir.

22 Q. Is the town named after the  
23 cigarettes or the cigarettes named after the  
24 town?

13:30 25 A. The name of the town came first.

51710 1185

13:30 1 Q. Okay. And it is here that RJR's  
2 main manufacturing facility is?

3 A. It is in this county.

4 Q. Okay.

13:31 5 A. Yes.

6 Q. Somebody told me that it's  
7 referred to as Tobacco City or something like  
8 that. Have you ever heard that word, that  
9 terminology?

13:31 10 A. I've never -- I've never -- I've  
11 never heard that. I mean, Winston and Salem  
12 cigarettes are manufactured here. I've never  
13 heard it called Tobacco City, though.

14 Q. Okay. Do you know how many  
13:31 15 cigarettes your company makes in a year?

16 A. I don't know the exact number, no,  
17 sir.

18 Q. Okay. If the 1991, I think,  
19 annual report said that y'all made about 110  
13:31 20 billion cigarettes here per year, would that  
21 seem correct to you?

22 A. That could be correct, yes, sir.

23 Q. Okay. Are all the cigarettes  
24 manufactured here sold to consumers in the  
13:32 25 United States?

51710 1186

13:32 1 A. I -- I don't know. At one time  
2 some cigarettes -- a few years back some  
3 cigarettes were manufactured here for sale in  
4 Japan. But I -- I don't know if that's still  
13:32 5 true or not.

6 Q. Okay.

7 A. I don't know.

8 Q. But at any rate, your company  
9 manufactures cigarettes here for sale in the  
13:32 10 United States certainly?

11 A. That's correct, yes, sir.

12 Q. And do you know whether or not  
13 cigarettes manufactured here in this county  
14 are sold in Texas?

13:32 15 A. I would be very much surprised if  
16 they're not. I don't see the billings or  
17 anything, but I would be very surprised if  
18 they are not.

19 Q. In your opinion, Doctor, are these  
13:32 20 cigarettes, the product that your company  
21 manufacturers and sells, safe?

22 A. The -- I don't know -- I don't  
23 know how to answer that. There are -- have  
24 been a number of -- of diseases, as you're  
13:33 25 well aware, that have been statistically

51710 1187

13:33 1 associated with the use of cigarettes. But  
2 then that's also true with the use of milk.  
3 So the risk associated with the use of  
4 cigarettes is well publicized.

13:33 5 Q. Okay. Does that -- I mean, in  
6 your opinion, is the product that your company  
7 sells a safe product to use as intended?

8 A. I used it -- I use the cigarette,  
9 I use the products that we manufacture, if  
13:33 10 that --

11 Q. Would you use it if you thought it  
12 was unsafe?

13 MS. FORBES: Just let the  
14 witness finish his answer, okay?

13:33 15 MR. THOMPSON: I will.

16 A. The -- I know that there are a  
17 number of epidemiological studies which have  
18 found an association of several different  
19 human diseases with the use of cigarettes.  
13:34 20 And yet I smoke cigarettes, I mean, I use  
21 them.

22 So there are risks associated with  
23 almost any human activity. And one man's safe  
24 might be another man's hazard. One of my sons  
13:34 25 walked in my house one afternoon and I asked

51710 1188

12:34 1 him where he had been and he said he had been  
2 skydiving. He paid \$100 for some guy to take  
3 him up and drop him out of an airplane at  
4 9,000 feet. I had a fit. His response, oh,  
13:35 5 it's safe.

6 Now, I understand that there are  
7 risks associated that have been -- through  
8 epidemiological studies have been found to be  
9 associated with the use of cigarettes. And  
13:35 10 yet, I use cigarettes. I smoke cigarettes.

11 Q. (By Mr. Thompson) Okay. And I  
12 don't want to try and beat it to death. But  
13 -- so you don't have an opinion as to whether  
14 or not the product your company sells is a  
13:35 15 safe product for the consumer?

16 A. I know that the -- that the  
17 product we sell has certain health risks  
18 associated with it established through  
19 epidemiology and that those health risks are  
13:35 20 well documented and advertised. The American  
21 public is well aware of those risks.

22 Q. Okay. And we spent some time  
23 earlier this morning talking about your  
24 opinion that it has not been established as  
13:35 25 fact that cigarette smoking, in fact, causes

51710 1189

13:36 1 these diseases in humans, correct?

2 A.--- To a scientific certainty, yes,  
3 sir.

4 Q. Right. Do you believe that your  
13:36 5 company has a responsibility to find out?

6 A. My -- my company, as any company,  
7 is responsible. They would -- we look at our  
8 products from a number of different ways  
9 constantly. If the product is broken, if  
13:36 10 there's something wrong with it and we can  
11 find it, we can fix it. We spend a lot of  
12 time looking at our products, trying to be  
13 sure we put the best tobaccos possible in our  
14 product, the cleanest processes and so forth.

13:36 15 And as I pointed out to you in our  
16 discussion this morning that -- my company has  
17 been responsive to the allegations about the  
18 risks associated with cigarette smoking in  
19 that over the past 40 to 50 years the company  
13:37 20 has -- has pioneered in the design of  
21 cigarettes through the use of filters, air  
22 dilution, special papers, blending. We have  
23 reduced the yields of these products from 33  
24 sales weighted down to 11 milligrams of tar  
13:37 25 sales weighted average. We pioneered in the

51710 1190

:37 1 development of cigarettes that don't burn  
2 tobacco and are still attempting to -- to  
3 introduce such a product into the  
4 marketplace.

13:37 5 So I see my company as very  
6 responsible.

7 Q. Having said all that then, is it a  
8 product that your company thinks is broken?

9 A. It is a product that my company  
13:37 10 knows a lot of other people believe is broken,  
11 but can't tell us what's broken about it. And  
12 consequently, as a responsible company, we've  
13 been trying to respond to allegations. And  
14 part of that is to reduce the yield of the  
13:38 15 product over the past 40 to 50 years.

16 Q. And that is what you see as  
17 your -- your responsibility to your consuming  
18 public?

19 A. We have -- we have a  
13:38 20 responsibility as a company to turn out the  
21 best possible cigarettes we can. And that's  
22 what we try to do.

23 Q. And when -- when your company  
24 makes statements about -- like you just made,  
13:38 25 the best possible cigarette, or we manufacture

51710 1191

13:38 1 a quality product, what does that mean?

2 A. That means that we pay close  
3 attention to all of the details in controlling  
4 the quality of that product. We put in good  
13:38 5 tobacco. We have -- we try to make a  
6 consistent product that meets our -- our  
7 customers' expectations. We try to satisfy  
8 the requirements of our customers.

9 Q. Don't you think, though, in making  
13:39 10 representations about quality products, best  
11 possible products, that there's an implication  
12 that this is a product that is safe to use as  
13 intended?

14 A. It's obvious from that question  
13:39 15 that you're not a smoker. The -- to a smoker,  
16 a quality product has -- and I am a smoker.  
17 It has certain connotations. One is that I  
18 don't get any surprises when I smoke a -- my  
19 cigarette, the one I happen to smoke, meets my  
13:40 20 expectations consistently. It doesn't have  
21 any off taste. It doesn't have any -- it  
22 doesn't have the bitterness associated with  
23 some cigarettes, and it's consistent from  
24 cigarette to cigarette. It doesn't have loose  
13:40 25 tobacco that falls out when I take the

51710 1192

1 cigarette out of the pack. It has consistency  
2 in that respect.

3 To say that I'm making a -- a  
4 quality cigarette or turning out a high  
13:40 5 quality cigarette would be sort of like I'm  
6 making very high quality ice cream. I mean,  
7 it's consistent, it has the taste that I want,  
8 and it meets my expectations.

9 Q. If your company was selling only  
13:41 10 to established smokers like yourself, however,  
11 you wouldn't be in business for very long,  
12 correct?

13 A. I don't know the answer to -- to  
14 that. I know that our main objective is to  
13:41 15 try to get people to switch to our products.  
16 I mean, we're -- that's where you're -- that's  
17 where your opportunity is. We are the  
18 second-ranked tobacco company. And we  
19 definitely would like to make end roads into  
13:41 20 our competition. These are people who already  
21 smoke cigarettes.

22 Q. Uh-huh. But certainly you're  
23 aware of -- of concerted efforts to address  
24 the new smoker market? I mean, that's clearly  
13:41 25 a goal of your company?

51710 1193

13:42 1 A. Well, if -- if there is a -- a  
2 pool of people out there that are going to  
3 begin to smoke that have made a decision or  
4 will make a decision to use cigarettes, we  
13:42 5 certainly would like to have them smoke our  
6 cigarettes if they're going to make that  
7 decision. But remember, you're asking .  
8 questions of a man who is a scientist and not  
9 a marketing person. So I -- I'm not an expert  
13:42 10 in what you're talking about.

11 I do know if you look at the --  
12 the information that's been published about  
13 the smoking market, our competitors -- we only  
14 have 25 percent of the market, about  
13:42 15 25 percent. That means that 75 percent of the  
16 market could be an opportunity for us. That's  
17 a lot of smokers. I think that's where we'd  
18 like to go is for people who are smokers.

19 Q. You have stated repeatedly today  
13:43 20 that it's your opinion that it has not been  
21 established that smoking causes any disease in  
22 human beings, correct?

23 A. To a scientific certainty --

24 Q. Okay.

13:43 25 A. -- that it is a cause of these

51710 1194

13:43 1 diseases, that's correct.

2 Q. Is there any disease that you  
3 believe smoking causes in a scientific sense?

4 A. I can't -- I can't say that I know  
13:43 5 of a disease that has been proved to be caused  
6 by smoking. Now, there may be. It may cause  
7 some of these diseases. Remember, I said  
8 that. But as far as the -- the scientific  
9 conclusion, I can't say that there is.

13:44 10 Q. And you also -- this morning we  
11 talked about the fact that based on that  
12 situation and your view of the evidence that,  
13 in fact, you smoke, you have chosen to smoke?

14 A. I didn't say it was based on that  
13:44 15 information.

16 Q. Okay.

17 A. I just said that I know the  
18 evidence. I know the allegations. I know the  
19 warnings. I know the -- you know, the history  
13:44 20 of the use of tobacco. But I -- I do smoke,  
21 yes.

22 Q. Uh-huh. So notwithstanding  
23 knowing all that you know, you've made a  
24 choice to smoke?

13:44 25 A. I smoke for whatever reasons. I

51710 1195

13:44 1 mean, you can say in spite of it or whatever.  
2 But I do smoke cigarettes. And I enjoy  
3 cigarettes.

4 Q. When you, as an expert on smoking  
13:45 5 and health or as director of smoking and  
6 health at RJR, makes a statement, a public  
7 statement that it has not been established .  
8 that smoking causes these diseases, in your  
9 opinion, is it reasonable for a layperson out  
13:45 10 in the consuming public to rely upon that  
11 statement?

12 A. In what sense?

13 Q. In making choices about whether to  
14 smoke or not.

13:45 15 A. I think -- I don't know how to --  
16 how to address the question. It is -- it's  
17 not made as a recommendation. I don't  
18 recommend that somebody start smoking. That's  
19 not what I do. I deal with the scientific  
13:46 20 evidence, and I try to be as objective as a  
21 can in stating, analyzing, and concluding from  
22 that evidence.

23 I don't know whether anyone would  
24 reasonably conclude that I had said it was  
13:46 25 okay to smoke or not. I mean, I have not

51710 1196

:46 1 discussed it with them. I don't know what  
2 their take-away is.

3 Q. But you don't think that the  
4 position that you're taking, your view of the  
13:46 5 evidence or your company's view of the  
6 evidence is an unreasonable view of the -- of  
7 the data, the facts that are out there, do  
8 you?

9 A. Oh, I think it's a -- I think the  
13:46 10 position is entirely consistent with the  
11 scientific evidence. And yet studies have  
12 shown that 95 or better percent of the -- of  
13 the American public believes that smoking  
14 causes these diseases.

13:47 15 So I'm not sure what your point  
16 is. I mean --

17 Q. I guess my point is is that if --  
18 if you and your company are going to great  
19 lengths in many cases to make your position  
13:47 20 known that we do not believe it has been  
21 established.

22 A. I don't think that at all. I  
23 don't think they have gone to great lengths to  
24 do it. I think we -- in the context of  
13:47 25 litigation, that has been our position. But

51710 1197

13:47 1 as far as actually going out on a PR campaign  
2 to -- to make that point to the American  
3 public, I don't see that we've actually done  
4 that.

13:47 5 Q. So -- I'm confused, because I  
6 don't understand -- is it your position that  
7 the consuming public ought to know the risks  
8 and, therefore, they are making a choice and  
9 if, in fact, it turns out, as you said it may,  
13:48 10 that smoking causes lung cancer, then they've  
11 just made a choice?

12 A. But that warning is on every pack  
13 of cigarettes that they smoke.

14 Q. Uh-huh.

13:48 15 A. I mean, the warnings have been  
16 mandated, they're on the -- on the  
17 cigarettes. And so the warnings -- and the  
18 warnings are well known. And the public, both  
19 the nonsmoking and smoking public, are well  
13:48 20 informed and well understand the risk  
21 associated with smoking, some of the most  
22 highly publicized health risks in the history  
23 of humankind --

24 Q. Uh-huh.

13:48 25 A. -- are the risks associated with

51710 1198

13:48 1 smoking.

2 Q. But it's your position and your  
3 company's position that that -- those are not  
4 the facts, that those positions that you're  
13:48 5 referring to are not accurate?

6 A. My position is that the risks  
7 associated with smoking are well known. I  
8 recognize those risks and will acknowledge  
9 that.

13:48 10 Q. Uh-huh.

11 A. What we have said is that the  
12 final proof, scientific conclusion that  
13 smoking actually causes these diseases has not  
14 come in. That final evidence has not come in.

13:49 15 Q. This morning we kind of briefly  
16 talked about the surgeon general's reports,  
17 and you referred to them. What is your  
18 understanding of the process by which those  
19 reports were generated? Of course, the first  
13:49 20 one was in 1964.

21 A. The -- my perception is this:  
22 That a contract is advertised, as I understand  
23 it, in the federal register for the production  
24 of a report. I believe that that is open for  
13:50 25 bids, I think that somebody will bid on

51710 1199

13:50 1 that -- that contract. And then it will be  
2 written under contract to the surgeon  
3 general. The surgeon general himself does  
4 not -- as I recall, does not write the  
13:50 5 report. But there are different people from  
6 different disciplines that participate in the  
7 writing of the report. That's my  
8 understanding.

9 Q. Okay. In 1989, the surgeon  
13:50 10 general issued a report that was a summary of  
11 the previous 25 years. And one of the  
12 conclusions in the 1989 report was that  
13 smoking is responsible for more than one in  
14 every six deaths in the United States and that  
13:51 15 smoking remains the single most important  
16 preventable cause of death in our society.

17 Do you agree with that statement?

18 A. I don't know whether the  
19 statement's true or not. The -- it may be.  
13:51 20 It may not be. I don't know whether it's true  
21 or not. I know the statement was made.

22 Q. You don't disagree with the  
23 statement?

24 A. As I said before, it has not been  
13:51 25 proved to my satisfaction that smoking

51710 1200

12:51 1 actually causes these diseases. Now, I know  
2 how he reached the conclusion that he reached  
3 based on -- on computations, calculations  
4 based on epidemiological evidence. And now,  
13:51 5 if smoking does cause these diseases at the  
6 rate in which he says it causes these  
7 diseases, then he may be correct. But once  
8 again, it may not be true. It may or may not  
9 be true.

13:52 10 Q. And -- what you're saying is we  
11 just don't know?

12 A. I'm saying that I don't know.

13 Q. And the same would be true then  
14 with the statements that were made and the  
13:52 15 conclusions reached in each of the surgeon  
16 general's reports simply because the  
17 methodology is something you don't agree with?

18 MS. FORBES: Wait just one  
19 second. I know we're not allowed to object.  
13:52 20 But are you asking him if he agrees with every  
21 statement in every surgeon general's report?  
22 Is that the question?

23 MR. THOMPSON: No.

24 MS. FORBES: Okay.

13:52 25 A. No, I understand -- I understand

51710 1201

13:52 1 that. What I'm -- what he's getting --

2 --- MR. THOMPSON: I'm going to  
3 shorten this deposition.

4 MS. FORBES: I will try not  
13:53 5 to do this. It is completely foreign to not  
6 object, but please don't ask that overbroad of  
7 a question.

8 A. The -- the conclusions with  
9 respect to causation, the conclusions with  
13:53 10 respect to the estimated mortality and so  
11 forth are conclusions that I do not give a  
12 blanket agreement to. I do not know whether  
13 smoking causes these diseases or not. It may  
14 cause these diseases, but I don't -- I can't  
13:53 15 prove that.

16 Q. (By Mr. Thompson) Do you believe  
17 nicotine is addictive?

18 A. Mr. Thompson, you're not talking  
19 to an expert on addiction. Now, I recall the  
13:54 20 definition of addiction in the 1964 surgeon  
21 general's report, and that by that definition  
22 it was not concluded to be an addictive  
23 substance. And it was only, I believe, in the  
24 1988 report in which the definition changed,  
13:54 25 did the surgeon general conclude that nicotine

51710 1202

11:54 1 was addicting. So it depends on which  
2 definition of addiction you're going to use.

3 Q. How about the 1988 definition?

4 A. By the 1988 definition, virtually  
13:54 5 everything you enjoy enough to do again is  
6 addicting.

7 Q. Okay. Certainly you'd agree with  
8 me, though, that nicotine is a drug?

9 A. Okay. What is your definition of  
13:55 10 a drug?

11 Q. Tell me what your definition is.  
12 That's simple.

13 A. There is -- from the  
14 pharmaceutical industry, there is a definition  
13:55 15 of a drug. And that is it depends on -- on  
16 what your claim is for the drug. Caffeine as  
17 it appears, for instance, in Diet Coke is not  
18 a drug. Caffeine as it appears in Nodol is a  
19 drug.

13:55 20 Q. How does that relate to cigarettes  
21 then?

22 A. Well, to my knowledge we make no  
23 claim about nicotine. I don't see -- nicotine  
24 is a native part of tobacco. It, of course,  
13:55 25 produces physiological response as does -- as

51710 1203

13:56 1 do many other things. But we make no claim  
2 about it."

3 Q. What do you mean, you make no  
4 claim about it?

13:56 5 A. I don't claim that it will keep  
6 you awake, that it will keep you asleep, it  
7 will help you lose weight. I don't make any  
8 claims about nicotine. It's part of the  
9 smoking pyridine. It's part of a cigarette.

13:56 10 Q. Does that affect whether it's a  
11 drug or not, under your own definition?

12 A. In that particular situation, I  
13 don't see it being defined as a drug by that  
14 particular definition of a drug.

13:57 15 Q. In some of your work at RJR you  
16 investigated nicotine analogs; is that  
17 correct?

18 A. The -- I had a -- earlier on I had  
19 a peripheral involvement with a program, a  
13:57 20 nicotine analog program. That was a number of  
21 years ago. That program has continued, I  
22 mean, there is a nicotine analog program  
23 within R & D that is focused toward a  
24 particular biological activity of nicotine  
13:57 25 that has been reported in the literature. And

51710 1204

:57 1 that is nicotine has been used in -- by a  
2 number of researchers, not only in the United  
3 States, but around the world, to attempt to  
4 treat short-term memory loss in Alzheimer's  
13:57 5 victims.

6 Q. Uh-huh.

7 A. The idea is -- with the analog, •  
8 can -- we'll go back to the structure activity  
9 relationship concept. Can I create a molecule  
13:58 10 that looks a little bit like nicotine that  
11 has -- is more potent in treating short-term  
12 memory loss. Do you understand what I'm --

13 Q. Uh-huh, I do.

14 A. So that's what -- this effect has  
13:58 15 been reported for Alzheimer's disease and  
16 Parkinson's disease. Whether it's real or  
17 not, I do not know. But I do know that other  
18 researchers have attempted to treat short-term  
19 memory loss -- I don't know about Parkinson's,  
13:58 20 but short-term memory loss and Alzheimer's  
21 with nicotine.

22 Q. But the idea is to take nicotine  
23 and create a synthetic version of it, an  
24 analog, that is altered so that it works  
13:59 25 better in the good ways and doesn't cause bad

51710 1205

13:59 1 things?

2 A. The idea would be, as with any  
3 chemotherapeutic agent, you want to  
4 eliminate -- what you want -- ideally what you  
13:59 5 would like to have is a chemical that does one  
6 thing and one thing only. Now, you're not  
7 going to achieve that. But you would like to  
8 pair off as much as possible any -- any  
9 biological activities that are not exactly  
13:59 10 what you want it to be.

11 In other words, I would like to  
12 really treat short-term memory loss;  
13 therefore, I don't want a compound that might  
14 increase heart rate or be a vasoconstrictor or  
13:59 15 whatever. I would like for it to do just that  
16 one thing.

17 In the case of Alzheimer's, my  
18 recollection is that when an Alzheimer's  
19 victim is being treated with nicotine, one  
14:00 20 encounters something called an anxiety --

21 Q. They get agitated, kind of?

22 A. It creates an anxiety of  
23 something, for whatever reason. I don't know  
24 why. We would like to see that side effect  
14:00 25 disappear and stay just with the short-term

51710 1206

11:00 1 memory loss.

2 Q. Has your company tried to do the  
3 same kind of process with your product, the  
4 cigarette?

14:00 5 A. I'm sorry?

6 Q. You've just described a process of  
7 taking a chemical, nicotine, and trying to  
8 eliminate or reduce the undesirable effects  
9 and maximize the desirable effects in the  
14:00 10 context of a pharmaceutical agent.

11 A. Using something like nicotine or  
12 something like that?

13 Q. Well, that process. Have you  
14 attempted to improve the cigarette product by  
14:01 15 doing the same thing?

16 A. Oh, if you look at the evolution  
17 of a cigarette, once again, I would  
18 recommend -- Dave Townsend knows --  
19 Dr. Townsend knows this story better than I  
14:01 20 do. But the fact is that we introduced things  
21 like porous paper and air dilution in order to  
22 reduce carbon monoxide yields in a cigarette.  
23 We have introduced various types of  
24 reconstituted sheet tobacco and puff tobacco  
14:01 25 along with filtration to reduce tar levels.

51710 1207

14:01 1 Q. Uh-huh.

2 A. So we have done that. And  
3 finally, we have attempted to introduce  
4 cigarettes that do not even burn tobacco. I'm  
14:01 5 not sure if this is answering the question you  
6 were getting. I mean, you asked --

7 Q. Well, I think so. In one hand, we  
8 were talking about your company's attempts  
9 to -- to, in effect, design a drug that does  
14:02 10 what you want it to do, but not what you don't  
11 want it to do?

12 A. Well, we would like to design a  
13 cigarette that addresses the allegations that  
14 have been leveled against our cigarettes. I  
14:02 15 mean, if that's what you're --

16 Q. Uh-huh.

17 A. And the only way we've been able  
18 to do that is to reduce the yields and finally  
19 to just try to produce a cigarette that  
14:02 20 doesn't burn tobacco at all.

21 Q. I think I found a statement that I  
22 think you will disagree with. The American  
23 Association for Cancer Research, AACR, are you  
24 familiar with the organization?

14:03 25 A. No.

51710 1208

14:03 1 Q. Okay. In their publication of  
2 April 1996 on lung cancer, they make the  
3 statement that "the most extraordinary feature  
4 of lung cancer is our knowledge of the  
14:03 5 causes. We know that smoking causes 85 to  
6 87 percent of all cases, 146,000 cases per  
7 year."

8 Do you agree with that statement?

9 A. I don't know that smoking causes  
14:03 10 lung cancer. As I said before, it may cause  
11 lung cancer.

12 Q. But any statement that says, we  
13 know that smoking causes a disease, you would  
14 disagree with?

14:03 15 A. I would disagree that I know that.

16 Q. Or that anybody knows?

17 A. Well, they can say what they want  
18 to about what they know. How can I know  
19 what's in their mind? They say that they know  
14:03 20 it. What I'm trying to tell you is what I  
21 know and what I don't know. I do not know, as  
22 a scientist, that smoking causes lung cancer.  
23 Now --

24 Q. But if another scientist were to  
14:04 25 say, I believe that it has been established, I

51710 1209

14:04 1 know that smoking causes lung cancer, then you  
2 wouldn't have a basis to disagree with that  
3 statement?

4 A. I would simply say that I don't  
14:04 5 agree with you.

6 Q. Okay. We've touched in general on  
7 some of the things that your company has done,  
8 and you've referred to product changes that  
9 have lowered -- I'm trying to paraphrase --  
14:05 10 lower the yield of tar and nicotine over the  
11 years. What kinds of changes were those that  
12 were made, and why were they made?

13 A. The -- oh, boy. Once again, now,  
14 I'm not the expert in this area. I know a  
14:05 15 little bit about it. I can talk to what I  
16 know with the caveat that I'm not an expert.  
17 But if you look back through the evolution of  
18 the cigarette as it appears today, you go from  
19 a cigarette that contained native tobacco and  
14:05 20 very little else. It was wrapped in paper.

21 Go back to the Camel cigarette  
22 that was introduced back in 1975. It was one  
23 of the first mass-produced cigarettes.

24 So you had tobacco wrapped in  
14:06 25 paper. Now, look at what's happened in --

51710 1210

:06 1 since -- excuse me, what did I say, '97 --  
2 1913. In the 80-odd years since then you've  
3 seen an evolution, one, in the paper. You've  
4 gone to paper that is porous, allowing gases  
14:06 5 to escape through the paper. You have seen  
6 the addition of filtration. Filters have been  
7 added to cigarettes.

8 Then you have seen those filters  
9 air diluted with perforations. Once again, it  
14:06 10 allows the escape of small molecule gases.  
11 You have seen the introduction of -- of  
12 reconstituted or synthetic sheet. In other  
13 words, tobacco that was unsuitable for  
14 cigarette manufacture because of its size or  
14:07 15 brittleness or something like that, could be  
16 made into a sheet, a paper sheet that could  
17 then be cut into cut filler that could be used  
18 in a cigarette.

19 You have seen the introduction of  
14:07 20 expanded tobacco, that is, tobacco that has  
21 been puffed by one process or another,  
22 expanded, so that you don't put as much  
23 tobacco into a cigarette. It now is less  
24 dense, takes up more space for less tobacco.  
14:07 25 You use less tobacco. Therefore, you can

51710 1211

14:07 1 reduce the amount of tar that's produced in  
2 that cigarette.

3 So all of those innovations and  
4 evolution of the cigarette have led to a  
14:08 5 reduction in the yield of the tar, nicotine,  
6 gases from the cigarette.

7 Q. You're familiar with, I guess,  
8 reports or controversy about the FTC method  
9 for testing the yield of cigarettes?

14:08 10 A. I am peripherally aware that that  
11 controversy has gone on, yes.

12 Q. Okay. And are you familiar with  
13 the concept of smoker compensation?

14 A. I have -- I have studied some of  
14:08 15 the papers that have been published on that,  
16 yes, I have.

17 Q. Okay. And do you have an opinion  
18 about whether or not smoker compensation  
19 offsets the reduced yields that you're talking  
14:08 20 about in the product?

21 A. Okay. In the absence of a lot of  
22 really good evidence, I'm -- it's hard to make  
23 concrete conclusions. I can tell you what I  
24 have read. Studies have been done -- that I  
14:09 25 have seen have been done in a number of

51710 1212

14:09 1 different countries including the United  
2 States... They've been done in Canada and Great  
3 Britain, the Netherlands. The issue has been  
4 addressed in some detail by the Froggett  
14:09 5 Commission in the UK. And one of the  
6 conclusions, one of the things that -- there's  
7 a couple of conclusions you can come away  
8 with. One, no two people smoke a cigarette  
9 the same way. That's one of the conclusions  
14:09 10 you're going to get immediately.

11 Q. Uh-huh.

12 A. If you have a thousand different  
13 people, then you're probably going to be  
14 looking at a thousand different ways to smoke  
14:10 15 a cigarette.

16 Q. Okay.

17 A. The British papers that have I  
18 seen refer to it as the smoking paradigm, a  
19 smoking maneuver. Not only that, you find  
14:10 20 that a certain percentage of people who smoke  
21 are noninhalers, don't inhale at all, which is  
22 sort of a surprise.

23 The Froggett Commission, rightly  
24 or wrongly, for whatever you know, whether you  
14:10 25 think they're right or they're wrong,

51710 1213

14:10 1 recommended -- and it was sort of a quasi  
2 government organization, made the  
3 recommendation that cigarette manufacturers  
4 make cigarettes in which the nicotine yield is  
14:11 5 fixed at a medium level, and that the tar  
6 yield is brought down against that fixed  
7 nicotine yield. Do you understand?

8 Q. I do.

9 A. Okay. In other words, their  
14:11 10 conclusion was that the health risks or the  
11 health hazards that we have found associated  
12 with cigarettes, we believe to come from the  
13 tar fraction. And that a smoker wants a  
14 certain amount of nicotine -- this is their  
14:11 15 conclusion -- from his cigarette. It may vary  
16 from smoker to smoker.

17 Therefore, if the amount of tar  
18 that is associated with that fixed amount of  
19 nicotine is reduced, you have reduced the dose  
14:12 20 of any potentially toxic materials there. Is  
21 that --

22 Q. Uh-huh.

23 A. That is a very crude summary of  
24 the Froggett Commission recommendation. Now,  
14:12 25 remember where that comes from. That comes

51710 1214

14:12 1 from the concept you just introduced to me of  
2 smoker-compensation. That is, if a smoker  
3 gets a cigarette, buys a cigarette with a low  
4 level of nicotine, he's going to compensate by  
14:12 5 smoking it harder, therefore, getting more  
6 tar.

7 Q. Uh-huh. Right. So that -- so •  
8 that the low yield cigarette, if the  
9 assumption is the smoker -- what the smoker is  
14:12 10 trying to get and what they are seeking  
11 delivery of is the nicotine, the smoker is  
12 going to compensate by smoking harder, faster,  
13 maybe more cigarettes and, in effect, end up  
14 with the same dose of nicotine and tar?

14:12 15 A. If that tar-to-nicotine ratio is  
16 the same, in other words, let's -- take two  
17 different cigarettes. And I'm -- I'm -- for  
18 the moment, let's just assume that they're  
19 correct. And the argument was that every  
14:13 20 smoker wants a certain amount of nicotine and  
21 some of them even said, every smoker wants one  
22 milligram of nicotine from his cigarette,  
23 which that's kind of hard to believe, but  
24 let's say you have a cigarette that yields 12  
14:13 25 milligrams of tar and one milligram of

51710 1215

14:13 1 nicotine. So the tar-to-nicotine ratio is  
2 12.

3 Compare that to a cigarette in  
4 which the tar is six milligrams and the  
14:13 5 nicotine is 0.5.

6 Q. Uh-huh.

7 A. The tar-to-nicotine ratio is still  
8 12. Now, their assumption is -- it's their  
9 assumption, that if he smokes -- he, the  
14:14 10 smoker, smokes his cigarette so that he gets  
11 one milligram of nicotine, smoking it harder,  
12 he's still got 12 milligrams of tar.

13 Q. Uh-huh.

14 A. Okay?

14:14 15 Q. Right.

16 A. Now, this is the -- the basis for  
17 their recommendation. This is -- this is  
18 their argument.

19 Q. The assumption of a linear  
14:14 20 relationship between tar and nicotine?

21 A. Well, they're saying if you've  
22 got -- yeah, that the -- the tar is fixed  
23 against the nicotine yield so that if I get --  
24 if the tar-to-nicotine ratio is -- is 12, then  
14:14 25 for every unit of nicotine I get, I'm going to

51710 1216

14:14 1 get 12 units of tar. And what they're saying  
2 is that if you fix the nicotine and reduce the  
3 tar against that nicotine, you can reduce --

4 Q. The tar?

14:14 5 A. -- the dose of tar that an  
6 individual gets. Now, that's the Froggett  
7 Commission, that's their argument. And they  
8 cited some evidence to that effect.

9 Q. Let me ask you just -- just for  
14:15 10 clarification, you're referring to the -- let  
11 me show you what I've marked as Simmons 2,  
12 which is Monograph 7 from the National  
13 Institute of Health, National Cancer  
14 Institute, which is a commission here in the  
14:15 15 United States.

16 (Deposition Exhibit 2  
17 was marked.)

18 MS. FORBES: Jeff, do you  
19 have copies for counsel?

14:15 20 MR. THOMPSON: I do, I'm  
21 sorry.

22 Q. (By Mr. Thompson) Have you  
23 reviewed this document as part of your review  
24 of scientific literature?

14:15 25 A. You know --

51710 1217

14:15 1 Q. And just for the record, I am not  
2 representing to you that that is the whole  
3 document. It is the cover page and kind of  
4 the forward.

14:15 5 A. Mr. Thompson, I don't believe -- I  
6 don't believe I have reviewed this document.

7 Q. Okay.

8 A. As a matter of fact, I don't see a  
9 date on this document.

14:16 10 Q. It is dated -- well, it does have  
11 a date. The question is, do I have the piece  
12 of it that has the date. Let me look real  
13 quick. I believe it's a 1996 document,  
14 though, Doctor.

14:16 15 A. It could be. It could be.

16 Q. I'm sorry. I don't.

17 A. I don't recall reviewing this  
18 document. I'm sorry, but I'm -- I'm not --

19 Q. Well, then it's unfair for us to  
14:16 20 talk about it. We will take it up later.

21 But at any rate, you're talking  
22 about the Froggett Commission?

23 A. Here, do you want this back?

24 Q. No, that's -- I've marked it.

14:17 25 MS. FORBES: It stays with

51710 1218

1 17 1 the court reporter's pile right there.

2 Q. (By Mr. Thompson) Just set it  
3 right there.

4 A. Okay. I got you. All right.

14:17 5 Q. You're at least aware of a  
6 commission, the Froggett Commission, which  
7 reached the conclusion that smokers compensate  
8 for the lower yield we've been discussing?

9 A. I believe I have a copy of that  
14:17 10 report, yes. I'm certainly aware of it and I  
11 have read the document and I'm almost certain  
12 to have a copy of it in my possession.

13 Q. And, of course, the -- the Premier  
14 project, which you and I touched on earlier,  
14:17 15 which was designed not to burn tobacco at all,  
16 but to simply heat it, would be kind of the --  
17 the -- the ultimate culmination of this --  
18 this direction that you --

19 A. Concept.

14:17 20 Q. -- and I have talked about.

21 A. There's a problem with it.

22 Q. What's that?

23 A. The -- it's because of the  
24 definition of "tar."

14:18 25 Q. Tell me, explain it for me.

51710 1219

14:18 1 A. For many years, in order to keep  
2 tobacco from drying out, from becoming really  
3 brittle and dry, the industry has employed  
4 glycerol as a humectant to keep the tobacco  
14:18 5 moist.

6 Q. Uh-huh.

7 A. Glycerol is a material that's  
8 found in a lot of pharmaceuticals, it's found  
9 in the human body. It's a pretty benign  
14:18 10 material.

11 Q. So it was added as an additive to  
12 the blend --

13 A. To keep it moist.

14 Q. -- to keep it moister?

14:18 15 A. Right.

16 Q. Okay.

17 A. Now, in the definition of tar, the  
18 FTC definition of tar, Federal Trade  
19 Commission definition, tar is equal to wet  
14:19 20 total particulate matter, WTPM, minus water --

21 Q. Okay.

22 A. -- and nicotine.

23 Q. Okay. So the water -- the  
24 retention of water affects the amount of tar?

14:19 25 A. Okay. Now, so what you do is when

51710 1220

14:19 1 you measure tar, you collect condensate on a  
2 pad, that's that brown material on a pad.

3 Q. Uh-huh.

4 A. You measure the amount of water  
14:19 5 and the amount of nicotine that's present.  
6 Subtract that from the total mass of material  
7 that's being captured.

8 Q. Okay.

9 A. And what is left is tar. But what  
14:20 10 is left also contains glycerol.

11 Q. Okay.

12 A. See, the idea was to just get at  
13 the material that is the --

14 Q. Incomplete products of combustion?

14:20 15 A. That's exactly right. But because  
16 of that definition, which occurred in the  
17 '60s, I believe, glycerol got captured in the  
18 definition of tar.

19 Q. Glycerol does not combust, it's  
14:20 20 not volatile?

21 A. Some of it may, but it is volatile  
22 and it comes through. In other words, it  
23 volatilizes and comes through as glycerol and  
24 is trapped on the pad as glycerol.

14:20 25 Q. Okay.

51710 1221

14:20 1 A. And therefore, when we made  
2 Premier, we used as the -- as the aerosol that  
3 simulates smoke -- are you familiar with what  
4 I'm --

14:20 5 Q. I am.

6 A. Okay. We used glycerol.

7 Q. Glycerol and what else?

8 A. We used glycerol.

9 Q. Just glycerol?

14:21 10 A. We put tobacco and glycerol inside  
11 the cigarette so that hot air came over it.  
12 The hot air picked up a lot of the flavor  
13 constituents from the tobacco, some nicotine,  
14 and it volatilized glycerol.

14:21 15 Q. Okay.

16 A. Now, when the glycerol comes  
17 through, it comes -- it volatilizes and all of  
18 the sudden it condenses and looks like smoke.

19 Q. Okay.

14:21 20 A. You --

21 Q. I do. I understand what you're  
22 saying.

23 A. Okay. It's like a ground fog. If  
24 you are out in the evening, you're driving  
14:21 25 along and you see -- it's not smoke that you

51710 1222

14:21 1 see, of course.

2 Q. Uh-huh.

3 A. It's water droplets in the air.

4 Well, in this case it's glycerol droplets.

14:21 5 Q. Plus, though -- I mean, if I  
6 understand the design correctly, you've got  
7 glycerol aerosol?

8 A. Right.

9 Q. Which is the, quote, smoke?

14:21 10 A. Right.

11 Q. And the flavor that is drawn out  
12 of the tobacco as --

13 A. And some nicotine that comes  
14 out --

14:22 15 Q. That comes out. So that is also  
16 in -- is it in a volatilized state or is it  
17 simply particulate?

18 A. No, what happens, most of the  
19 flavor compounds that come out of tobacco will  
14:22 20 dissolve in a glycerol droplet so they will be  
21 caught on the pad. If you look at the pad,  
22 though, that you smoke, it will be white.

23 Q. Okay.

24 A. No brown material there, so you've  
14:22 25 virtually eliminated the products of

51710 1223

14:22 1 incomplete combustion of tobacco.

2 Q. But you still have the glycerol?

3 A. Still have the glycerol.

4 Q. So it's still considered tar?

14:22 5 A. And because -- that's right.

6 Because -- now, let's calculate the tar on  
7 this pad. So what do we do? We weigh the  
8 pad. We know what the pad weighed when we  
9 started.

14:22 10 Q. Uh-huh.

11 A. Let's say it weighed a gram. And  
12 then we weigh it after we put it through and  
13 it weighs one and a half grams. Okay. I  
14 measure a tenth of a gram of water, a tenth of  
15 a gram of nicotine, and now I'm left with  
16 three-tenths of a gram of tar, which is the  
17 glycerol.

18 Q. So to successfully take out what  
19 you wanted to take out --

14:23 20 A. But because of the definition --  
21 that's right.

22 Q. Because of the definition of it,  
23 you were still technically saddled with tar?

24 A. That's right.

14:23 25 MS. FORBES: Dr. Simmons,

51710 1224

14:23 1 just for the record and making the court  
2 reporter's job easy, let counsel let his  
3 question out and then you talk.

14:23 4 THE WITNESS: I beg your  
5 pardon.

6 MR. THOMPSON: I am also  
7 doing it. Part of it is I'm trying to keep it  
8 going so we can get out of here, and I  
9 apologize for interrupting you.

14:23 10 Q. (By Mr. Thompson) If I understood  
11 your testimony from this morning correctly,  
12 however, the reason the product ultimately was  
13 not successfully marketed was the consumers  
14 didn't like it?

14:23 15 A. It did not -- it did not catch on  
16 at the marketplace. There were -- there  
17 were -- the problem was that it didn't burn  
18 back like a cigarette does. It -- when you  
19 got through smoking, it was the same length as  
14:24 20 when you started, all right?

21 Q. It didn't go away?

22 A. So there was no cue that this  
23 cigarette's out, I can put it out. The cues  
24 that a smoker normally gets were not there,  
14:24 25 all right? Plus the fact, it had an aroma to

51710 1225

14:24 1 it that was very displeasing to a smoker.

2 Now, a lot of people got used to it.

3 Q. Uh-huh.

4 A. But it took a long time to get

14:24 5 used to it. It smelled sort of like baking

6 bread. I heard it described like baking

7 bread. Well, that's not like a cigarette, •

8 okay? So it didn't meet the smoker's

9 expectations, and it didn't catch on at the

14:24 10 marketplace.

11 Q. But if I've understood kind of

12 this whole progression, irregardless of the

13 fact that the evidence has not been clear

14 about exactly how and why biologic activity

14:25 15 occurs in cigarette smoke, it seems clear to

16 me and y'all and everybody knows that it is

17 the incomplete products of combustion that are

18 the problem, if there's a problem?

19 A. This is the -- the prevailing

14:25 20 opinion throughout the community is that the

21 vast majority of any health risks are going to

22 associated with the tar fraction. Now,

23 there's a -- there's a group out there that

24 talks about carbon monoxide, too, I don't want

14:25 25 to mislead you. Carbon monoxide is in

51710 1226

1 25 1 cigarettes, it was in Premier. You know, we  
2 said it was there. We said how much was  
3 there. It's in Eclipse. We're still working  
4 to try to get it down. I mean, but my -- you  
14:25 5 know, my take-away from conversations with  
6 members of the community is that the -- the  
7 tar is the thing that needs to be reduced in  
8 cigarettes in order to address the health  
9 risks associated with cigarettes, that's  
14:26 10 correct, yes.

11 Q. And the Premier project  
12 successfully did that in terms of the science?

13 A. That's correct.

14 Q. But the product was ultimately  
14:26 15 taken off the market because commercially it  
16 was not successful?

17 A. That's correct.

18 Q. Do you need to take a break?  
19 That's fine.

14:26 20 A. I'm over 60 years old now.

21 THE VIDEOGRAPHER: Off the  
22 video record.

23 (A recess was taken.)

24 THE VIDEOGRAPHER: We're on  
14:34 25 the video record.

51710 1227

14:34 1 Q. (By Mr. Thompson) Let's keep  
2 moving. Doctor, we've talked about a lot of  
3 different things, and we've talked about kind  
4 of the development of the product. We were  
14:35 5 just discussing the Premier cigarette.

6 (Deposition Exhibit 3  
7 was marked.)

8 Q. Let me show what I've marked as  
9 Exhibit 3, which is a copy of an ad I read on  
14:35 10 the plane the other day. What's this all  
11 about?

12 A. You want to be a little more  
13 specific?

14 Q. I knew she couldn't object, so I  
14:35 15 had to ask that question. How is it that  
16 removing additives is something that's  
17 perceived as good at your company?

18 A. By whom?

19 Q. Well, in your opinion, is it  
14:35 20 something that's good?

21 A. It goes -- it goes something like  
22 this. For many years there were a lot of  
23 statements made in the press, and we don't  
24 know what's in cigarettes. The implication  
14:36 25 was to the public, the public perception was

51710 1228

14:36 1 that there were things that we put in  
2 cigarettes that were bad. We attempted to  
3 answer that allegation by making all of the  
4 additives industry-wide public, publish a  
14:36 5 document which contained all of those  
6 additives.

7 Q. That was in 1994, correct?

8 A. Mr. Thompson, the dates -- I don't  
9 recall the dates. But that could be very well  
14:36 10 correct. It was about that time.

11 (Deposition Exhibit 4  
12 was marked.)

13 Q. Let me, just so we get our dates  
14 straight, show you what's marked as  
14:37 15 Exhibit 4. And it's a series -- it's not just  
16 a Philip Morris document, there are actually a  
17 couple of -- there's an AP wire story as  
18 well. I think it mentions your -- the second  
19 article is really the one that mentions RJR.

14:37 20 MS. FORBES: Again, if I  
21 could have a copy of the exhibit, please.

22 MR. THOMPSON: I'm sorry. I  
23 thought I handed you one. I was trying to be  
24 sneaky.

14:37 25 MS. FORBES: Got to watch

51710 1229

14:37 1 it.

2 Q. (By Mr. Thompson) And I hand you  
3 this only to see if it will refresh your  
4 recollection as to the date.

14:37 5 A. That looks about correct. I mean,  
6 I -- I have seen a copy of the industry-wide  
7 ingredients package. That ingredients  
8 package, or that list of ingredients had been  
9 evaluated by some toxicologist who wrote an  
14:38 10 opinion on it. The ingredients were, for the  
11 most part, listed as -- generally regarded as  
12 safe or accepted for use in foods and as  
13 additives in a number of things.

14 Q. And you've already said you're not  
14:38 15 an expert on the issue of additives and those  
16 issues; is that correct?

17 A. That's correct, yeah.

18 Q. Because I don't want to get into  
19 GRAS and FDA approval and all those things if  
14:38 20 we don't have to.

21 A. Not a problem.

22 Q. You're not going to offer any  
23 opinions about those issues at trial, are you?

24 A. I'm not an expert. I'm not --  
14:38 25 that's somebody else.

51710 1230

14:38 1 Q. That's fine.

2 A. The public -- it's my  
3 understanding that our read of the public or  
4 our understanding about the public was that  
14:38 5 ingredients had been raised to an issue with  
6 them. And, therefore, we elected to create a  
7 cigarette that really didn't have any  
8 ingredients at all. And that is -- that's my  
9 understanding of what this is all about. We  
14:39 10 have just taken the ingredients out of Winston  
11 cigarettes.

12 Q. And it's your understanding that  
13 was done more to address a public perception  
14 than to address smoking and health concerns?

14:39 15 A. It was done to -- as -- yes,  
16 that's correct. It was not -- not from the  
17 standpoint that we believe that the  
18 ingredients were causing some problem, some  
19 health problem, but because the public  
14:39 20 perceived that they might be a problem. So we  
21 took them out.

22 Q. Were any of -- in the previous  
23 products that had ingredients in them -- not  
24 talking about the Noble cigarettes, the  
14:40 25 conventional cigarettes -- were any of the

51710 1231

14:40 1 incomplete products of combustion that we've  
2 talked about previously the product of  
3 pyrolysis of the additives?

4 A. Okay. Once again, now, you're not  
14:40 5 talking to an expert on this. But one of the  
6 things that I can tell you which has been  
7 published which I have read which I understand  
8 is that there are some materials that are in  
9 the ingredients in one way or another when  
14:41 10 they combust, when they -- excuse me, when  
11 they pyrolyze as opposed to burn --

12 Q. Uh-huh.

13 A. -- produce flavor compounds. Now,  
14 these flavor compounds have been evaluated for  
14:41 15 their toxicity, for their biological activity  
16 and are known to be benign. They are used,  
17 for instance, in the food industry.

18 Q. Uh-huh.

19 A. For example, there are things  
14:41 20 called pyroxenes which you find -- some of  
21 them will produce a chocolate-like aroma,  
22 some, a peanut-like aroma. You might get a  
23 lot of different flavor notes out of perosenes  
24 and they are utilized in the food industry.  
14:41 25 Since they are volatile, you can't put a

51710 1232

perosene in a cigarette. You put in something that will pyrolyze into the perosenes, okay, and --

Q. And the perosene is what was tested?

A. And the perosenes have been tested for -- in addition to the precursor to the -- to the perosenes. In other words, if an amino sugar is the precursor to the perosene, the amino sugar has been tested because it was used in the food industry. And then the perosene, which will appear on cooking, has been tested also.

Q. Uh-huh.

A. You find a lot of perosenes and related molecules in coffee and in chocolate and things like this. They produce that type of flavor and taste and so forth.

Q. If I understand the ingredients release correctly, however, it is a listing -- a listing of all the ingredients used in cigarette products, and there is no way that I can go find out which specific ingredients are in a specific cigarette or specific brand of cigarettes?

51710 1233

14:43 1 A. I believe that's correct. I  
2 believe that that is a listing of ingredients  
3 which represents a summation of everything  
4 used in the cigarettes manufactured in the  
14:43 5 United States. That's my understanding.

6 Q. So as a consumer, if I were a  
7 smoker, I still couldn't go find out what was  
8 in Winston Lights or whatever specific  
9 cigarette brand I smoked?

14:43 10 A. I believe that's the case.

11 Q. Why did it take until 1994 for the  
12 tobacco industry to release this information?

13 A. The information -- until when?  
14 Excuse me?

14:43 15 Q. 1994.

16 A. Once again, I'm not the expert in  
17 this area. But the list had been in the hands  
18 of the Department of Health, Education and  
19 Welfare, HEW, for many years preceding that,  
14:43 20 as I understand, Mr. Thompson. I don't recall  
21 when it was first placed in their hands.

22 Q. True, but subject to a prohibition  
23 and making it public, they did not have -- the  
24 ingredients were provided to HHS with the  
14:44 25 understanding that it would not be made

51710 1234

14:44 1 public?

2 A. I think that's -- that's right.  
3 It could be evaluated by the HHS.

4 Q. Right. And so my question was  
14:44 5 intended to be focused on the information  
6 being provided to the consuming public.

7 A. I know that we're in an awfully  
8 competitive industry, and that this is --  
9 that's considered generally -- it's not even  
14:44 10 patented information. That's considered to be  
11 proprietary information, and I would like to  
12 know what makes a Marlboro cigarette a  
13 Marlboro cigarette, for example.

14 Q. I understand. The FDA has  
14:45 15 recently taken some steps or announced some  
16 steps to reduce youth access to cigarettes.  
17 You're aware of that?

18 A. I'm aware that for a number of  
19 years there has been a campaign in the United  
14:45 20 States, if not elsewhere, but certainly in the  
21 United States, to limit youth smoking; to  
22 eliminate underage smoking, certainly.

23 Q. Do you agree that that's a good  
24 thing to do?

14:45 25 A. I certainly do.

51710 1235

14:45 1 Q. Why?

2 A. Smoking, like drinking and other  
3 things, are adult customs. They are for  
4 adults. Underage people are not adults. They  
14:45 5 should not indulge in adult customs. I mean,  
6 this is the way I was brought up. I'm not  
7 sure what your upbringing was. But I was not  
8 even allowed to drink coffee until I was in  
9 high school, but that's me.

14:46 10 Q. Are you aware of your company ever  
11 targeting youth in marketing?

12 A. I am not at all. And -- but once  
13 again, I think the people you should direct  
14 questions like that to would be in the  
14:46 15 marketing area. I'm not a marketing person.  
16 I wouldn't -- I just don't know about  
17 marketing.

18 Q. Okay. But at least in your own  
19 knowledge, you don't have any personal  
14:46 20 knowledge of specific attempts to target  
21 youth?

22 A. No, sir, I do not.

23 Q. Do you have any knowledge of  
24 specific programs or intent to target Blacks  
14:46 25 or Hispanics or other minority groups?

51710 1236

1 :46 1 A. We manufactured a cigarette at one  
2 time for -- called Uptown that was designed to  
3 meet what we perceived to be some  
4 expectations. The reason I know this, I sat  
14:47 5 in on an explanation of the thing one time in  
6 which a study had been done for intercity  
7 populations, which in certain cases were  
8 primarily Black populations. And the name of  
9 the cigarette was Uptown. I mean, you're no  
14:47 10 doubt aware of this.

11 Now, it was advertised in Black  
12 neighborhoods. It had a marketing twist to  
13 it, which we perceived, or at least I think  
14 our marketing department perceived as being  
14:47 15 something that was desired by the smoking  
16 community, that particular smoking community.

17 Now, I can't say that it was  
18 targeted toward Blacks, but I think that was  
19 the -- the take-away or a lot of people  
14:48 20 criticized it that way.

21 Q. Uh-huh.

22 A. It was very simply that the  
23 cigarette was put in the pack upside down  
24 against the way it would normally be in so  
14:48 25 that people who work with their hands,

51710 1237

14:48 1 mechanics or people who were working manually  
2 when they take a cigarette out, they don't  
3 have to touch the filter end that goes in  
4 their mouth, they could just pick it up by the  
14:48 5 non-filter end and put it in their mouth so  
6 they don't have to do that. And that's all  
7 there was to it.

8 Q. Uh-huh.

9 A. But we pulled it from the market.

14:48 10 Q. Okay.

11 MS. FORBES: Jeff, as I  
12 indicated, I need to make a quick phone call.

13 MR. THOMPSON: Sure. Go off  
14 the record.

14:48 15 THE VIDEOGRAPHER: Off the  
16 video record.

17 (A recess was taken.)

18 THE VIDEOGRAPHER: We're on  
19 the video record.

14:50 20 Q. (By Mr. Thompson) Okay. We're  
21 back. We were talking a little bit about  
22 marketing issues.

23 Doctor, do you have any knowledge  
24 or an understanding of what demographically is  
14:50 25 the -- the typical smoker of your company's

51710 1238

11:50 1 products?

2 A. No, sir, I don't.

3 Q. Okay. Would it surprise you if --  
4 if I told you that it is typically

14:50 5 lower-educated working class, in some cases,  
6 minorities?

7 A. I wouldn't know how to react. I  
8 wouldn't be surprised. I mean, that's --

9 Q. Okay. Let's talk a minute about  
14:51 10 scientific integrity. You've -- you've -- you  
11 are the principal -- a principal scientist at  
12 RJR?

13 A. Right.

14 Q. We've talked about your view of  
14:51 15 the scientific evidence and your view that it  
16 is not conclusive on the issue of smoking and  
17 health. Do you know -- do you have any  
18 personal knowledge of whether your company has  
19 ever withheld information about its product  
14:51 20 from the government or from the public that  
21 relate to those issues?

22 A. I have no knowledge that they have  
23 ever withheld information that would bear on  
24 that, no, I don't.

14:52 25 Q. And in the context of your -- our

51710 1239

14:52 1 discussion about trying to find the answer,  
2 trying to find the truth, that would clearly  
3 be inappropriate conduct; would you agree?

14:52 4 A. I'm sorry? Would you ask the  
5 question again, please?

6 Q. In the context of the scientific  
7 inquiry about trying to determine whether or-  
8 not smoking causes diseases of a human animal,  
9 it would be inappropriate for a company to  
14:52 10 withhold information that might relate to  
11 smoking and health?

12 A. I believe valid scientific  
13 information should be published in accordance  
14 with good scientific principle and peer review  
14:52 15 journals, if that's your question. It should  
16 be -- if you have valid scientific  
17 information, then you publish it.

18 Q. Let's take a look at what I have  
19 marked as Exhibit 1 real quick. You've got it  
14:53 20 right there.

21 We've talked a lot about a lot of  
22 issues. And what I would like to do is kind  
23 of go through Exhibit 1 real quickly and make  
24 sure that I have an understanding of the  
14:53 25 opinions that you've been asked to formulate

51710 1240

:53 1 and offer in this case.

2 The first paragraph is something  
3 we've talked in and out of all day. It is --  
4 well, if you will, just -- can you explain to  
14:53 5 me what the -- what the -- your primary point  
6 is with regard to Paragraph 1?

7 A. The lines of evidence that one  
8 tries to acquire or to evaluate would be one  
9 of an epidemiology nature, toxicology, which  
14:54 10 would be certain types of studies in animals,  
11 chemical information; that is, identity of a  
12 chemical, its behavior, and a mechanism by  
13 which any of these things might initiate or  
14 propagate some chronic disease.

14:54 15 Now, in this particular area, the  
16 ideal -- in order to establish causation, your  
17 ideal piece of information is a mechanism by  
18 which some chemical initiates and propagates a  
19 process. In the absence of that, then you  
14:54 20 have to look at other types of information.  
21 And you like for that information to converge.

22 Q. Okay. Let me stop you for a  
23 second. You've said that, A, finding the  
24 specific mechanism of cause and effect is the  
14:54 25 ideal situation?

51710 1241

14:54 1 A. That is the one that would be  
2 ideal, that's correct.

3 Q. And when you refer to the specific  
4 mechanism, are you referring to a specific  
14:55 5 observation, if you will, of -- of an exposure  
6 and effect in a human?

7 A. When I'm talking about a  
8 mechanism, I'm talking about something that  
9 occurs at the molecular level. I understand  
14:55 10 the molecular larger events which transpire to  
11 create this chronic disease.

12 Q. Uh-huh. Okay.

13 A. All right. An example would be  
14 the -- one of the current theories for cancer  
14:55 15 is the so-called oncogene theory in which it  
16 is believed that all of us in our nuclear DNA  
17 carry one or more copies of some oncogene or  
18 the other. In other words, a cancer-causing  
19 gene, which is repressed, it's not active.  
14:56 20 It's not allowed to express itself.

21 Q. Uh-huh.

22 A. But some circumstances might arise  
23 in which through either a chemical exposure or  
24 a radiation exposure, something, might allow  
14:56 25 that gene to express itself, thereby

51710 1242

14:56 1 triggering a change in the identity of the  
2 cell in which that nucleus is contained,  
3 creating a wild cell, which then begins to  
4 propagate as neoplasia and goes and then  
14:56 5 metastasizes and then becomes cancer. I mean,  
6 this is a progression of events. Ideally, one  
7 day we will have that. Prog --

8 Q. Give -- I'm sorry, go ahead.

9 A. A process is under way now in the  
14:56 10 United States and perhaps around the world to  
11 map the entire human genome. You may be  
12 aware.

13 Q. Uh-huh, I am.

14 A. Ambitious.

14:57 15 Q. Yes.

16 A. But costly, but it will be done.  
17 And that will lead to a lot of understanding  
18 not only of the oncogene, but the tumor  
19 suppressor genes, the genes that keep the  
14:57 20 oncogene under suppression.

21 Q. Uh-huh.

22 A. We may, through something like  
23 that, get at the mechanism. May not. There  
24 may be other ways that we get at the  
14:57 25 mechanism. But that is what I mean when I say

51710 1243

14:57 1 mechanism. There is maybe some chemical  
2 agent, maybe some physical agent, may --  
3 something else may release or derepress that  
4 gene so it can express itself as -- change the  
14:57 5 identity of the cell and convert into a cancer  
6 cell.

7 Q. So in the context of smoking and  
8 health then, smoking and cancer, the ideal  
9 piece of the puzzle, the ideal piece of  
14:58 10 evidence would be exposure to cigarette  
11 smoke? I mean, the --

12 A. Or some component of cigarette  
13 smoke.

14 Q. Okay.

14:58 15 A. I mean, as it appears in cigarette  
16 smoke. In other words, we know, as you  
17 alluded to earlier and people have published,  
18 there are a lot of things in cigarette smoke.  
19 Tar is -- there is not a molecule called tar,  
14:58 20 okay. How does this thing -- and even  
21 recently, Sir Richard Dahl has said we don't  
22 know what thing or things in cigarette smoke  
23 actually cause these diseases.

24 Q. Uh-huh.

14:58 25 A. We don't.

51710 1244

14:58

1 Q. Right.

2 A. But if -- for the mechanism you  
3 would have to have a component that initiates  
4 this process by perhaps by derepressing an  
14:59 5 oncogene in some way so that the cell could  
6 then express itself a different way. That  
7 oncogene converts the identity of a cell. •

14:59

8 Q. Some change in the genetic makeup  
9 of a human cell that causes either this  
10 oncogene to allow to -- to express itself --

11 A. Uh-huh.

12 Q. -- or a change that might cause  
13 the suppressor gene to stop --

14 A. Repressor.

14:59

15 Q. -- preventing it from expressing  
16 itself?

17 A. That's right. These are theories  
18 that are on the table, that's correct.

14:59

19 Q. Uh-huh. That evidence, if it were  
20 available, even in the absence of an animal  
21 model, would that satisfy your -- your  
22 requirement of scientific proof?

23 A. The -- you've got to remember, I  
24 don't want to see this done in a test tube.

15:00

25 Q. Uh-huh.

51710 1245

15:00 1 A. I want to see this done in a -- a  
2 system that has all of the defense mechanisms  
3 intact. Remember, there are enzymes that --  
4 biochemistry is not very creative when it  
15:00 5 comes to naming enzymes, repair enzyme.

6 Q. Uh-huh.

7 A. Okay. These enzymes will detect  
8 damage in DNA and repair that damage.

9 Q. Uh-huh.

15:00 10 A. So I want to see this mechanism  
11 exist in an organism which has its different  
12 mechanisms intact.

13 Q. Ideally in a human being?

14 A. The human being is not a good  
15:00 15 experimental animal.

16 Q. And I didn't mean to imply that.

17 A. I mean, I -- I wouldn't -- and I  
18 understand that. You're not talking about  
19 doing an experiment on a human being. But I  
15:00 20 would like to see it done so that I can  
21 accurately and confidently translate that  
22 event to something that would occur in a human  
23 being.

24 Q. But if you could observe that  
15:01 25 event in a human being, that would be the

51710 1246

1 01 1 ideal evidence?

2 A. Once again, that's hypothetical.  
3 It would be awfully good.

4 Q. Okay. Would it change your mind?

15:01 5 A. Depending on the quality of the  
6 evidence, it could.

7 Q. Hypothetically speaking, if  
8 tomorrow you -- somebody handed to you or they  
9 said, Doctor, fly out here and let's -- I want  
15:01 10 to show you something, and somebody presented  
11 to you the ideal piece of evidence, whatever  
12 it is, and you make that call, what would  
13 your -- in your opinion, what would your  
14 recommendation be to your employer?

15:01 15 A. This would be happy hour. What  
16 I've got now is a mechanism and a trigger.  
17 Now I have a specific target for product  
18 development, product design.

19 Q. Uh-huh.

15:02 20 A. I have something I can directly  
21 attack.

22 Q. As a scientist?

23 A. As a -- yeah, as a scientist and  
24 as a product developer.

15:02 25 Q. Uh-huh.

51710 1247

15:02 1 A. I can go in, I've got it, I've  
2 identified it, now I can fix it.

3 Q. Uh-huh.

4 A. See, what you're saying to me is  
15:02 5 somebody is coming in and is going to show me  
6 exactly what's broken.

7 Q. That's right.

8 A. And at that point I have a target  
9 that I can fix.

15:02 10 Q. Would you recommend that the  
11 company stop selling the product until you fix  
12 it?

13 A. I would recommend that the company  
14 make notice and fix it. I wouldn't say stop.  
15:02 15 I would just say, look, this is a -- this is a  
16 different decision. I wouldn't make a  
17 recommendation like that. I would just simply  
18 say, look, here's the evidence, I'm convinced  
19 we're going to have to fix this.

15:03 20 Q. Uh-huh.

21 A. And then do it.

22 Q. Okay. And in -- in the meantime,  
23 it's okay to sell -- continue selling the  
24 cigarettes until they're fixed?

15:03 25 A. As long as the -- the notification

51710 1248

15:03 1 is there. I mean, you're not -- you would --  
2 you would say, look, this piece of evidence is  
3 out here, here is a mechanism, we are  
4 convinced that this is correct, we're going to  
15:03 5 fix it. In the meantime, be advised.

6 Q. So in your opinion, it would be  
7 acceptable to continue selling the product? \*

8 A. I would continue to smoke it.

9 Q. That's a different question.

15:04 10 A. Look at it this way. In the --  
11 take the alcohol industry. One of the things  
12 that just about everybody agrees on is that  
13 alcohol is addictive and has been known to  
14 destroy families on top of families. Is it  
15:04 15 irresponsible to not sell it, to stop selling  
16 it? I don't think so. People are aware of  
17 the risk associated with consuming alcohol.  
18 They are aware of the fact that many  
19 automobile accidents and other types of  
15:04 20 disasters are triggered by people who consume  
21 alcohol. Is it irresponsible for the alcohol  
22 industry to continue to sell beer or  
23 whatever? I don't think so.

24 Q. Somebody's going to do it, so --

15:05 25 A. I mean, we went through this. We

51710 1249

15:05 1 had prohibition.

2 Q. Uh-huh.

3 A. Did it work? No, sir, it didn't  
4 work.

15:05 5 I don't see it as irresponsible,  
6 no.

7 Q. Mechanism was the first --  
8 actually it's the fourth, but we talked about  
9 it first. And it is your opinion that to  
10 date, there is no evidence of the mechanism?

11 A. I have not seen a mechanism --  
12 I've seen theories of mechanism, but I have  
13 not seen one tested all the way that showed it  
14 to be true.

15:05 15 Q. Okay. Let's start at Number 1  
16 again. The epidemiology we have talked in and  
17 out of. In terms of epidemiology, are you  
18 satisfied that the epidemiologic evidence  
19 supports not only an association, but a very  
15:06 20 strong association between smoking and a  
21 number of health -- serious health problems?

22 A. The epidemiology that I have seen,  
23 while it is not entirely consistent, shows a  
24 strong association between smoking and a  
15:06 25 number of diseases, yes, that's true.

51710 1250

15:06 1 Q. And that includes lung cancer?  
2 A. Cardiovascular disease.  
3 Q. Cardiovascular disease, cancers of  
4 the head and neck?  
15:06 5 A. Emphysema.  
6 Q. Emphysema? Okay. You have  
7 mentioned and just -- I think I have read it  
8 in a previous depo about gaps in the  
9 epidemiologic evidence and inconsistencies. I  
15:06 10 don't want to spend hours going through that.  
11 But I think the quickest way to do it is for  
12 me to show you something, if I can find it.  
13 (Deposition Exhibit 5  
14 was marked.)  
15:07 15 Q. Let me show you what I have marked  
16 as Simmons Number 5. And before you look at  
17 that, we previously discussed real briefly  
18 some two new studies that you had heard of  
19 that relate to nutrition and cancer which  
15:07 20 are -- or I guess which would be data that, I  
21 guess, would fall into the category of kind of  
22 confounding or inconsistent data.  
23 A. I don't know what it's going to  
24 show. I don't want to try to make too much  
15:07 25 out of it. Here is the blurb that I have seen

51710 1251

15:07 1 on it; that is, the brief write-up goes like  
2 this: A report will be coming out of a quasi  
3 governmental group in the United Kingdom and  
4 some organization associated with -- a second  
15:08 5 report out of some organization associated  
6 with the World Health Organization, two  
7 different reports.

8 Q. Uh-huh.

9 A. According to the write-up that I  
15:08 10 saw, they both are going to reach about the  
11 same conclusion. And that is, a high  
12 percentage of cancers are going to be  
13 associated with nutrition.

14 Q. Okay.

15:08 15 A. I wish I had more on them. I  
16 don't know whether -- what they base this on.  
17 See, I don't even know what evidence they're  
18 using to make these claims. I don't -- I  
19 don't think -- my conclusion is -- or my  
15:08 20 take-away is that these are not epidemiology  
21 studies that they're looking at. It's  
22 something else. Some other type of evidence.  
23 I don't think they've done an epidemiology  
24 study on this.

15:08 25 Q. Okay. Well, we will take a look

51710 1252

15:08 1 at it. Tell me, if you can, in terms of the  
2 epidemiology and the inconsistencies or gaps  
3 what are the top concerns that you can think  
4 of in the epidemiologic record. If I said,  
15:09 5 give me your top five or the top three, what  
6 are they?

7 A. Okay. The problems with  
8 epidemiology go like this with the  
9 epidemiological --

15:09 10 Q. I am going to interrupt you just  
11 because I want to try and -- I think we've  
12 discussed kind of the issue of epidemiology  
13 versus causation and inference versus  
14 causation. What I would be interested in is  
15:09 15 if you've got some specific instances where  
16 you think there is -- you know, there are  
17 studies or there are gaps in the -- in the --  
18 the record --

19 A. I'm not sure what you're talking  
15:09 20 about.

21 Q. -- that confuse the issue. Well,  
22 then, go ahead. I'm sorry.

23 A. I don't -- I'm not trying to  
24 resist your question. I just don't understand  
15:09 25 the question. What I'm trying to get at is

51710 1253

15:09 1 that when I look at an epidemiology study --  
2 and I've looked at the ones on smoking and,  
3 say, lung cancer and different types of  
4 disease.

15:10 5 Q. Uh-huh.

6 A. If you -- if I do an epidemiology  
7 study today, I start one, now, what I want to  
8 do is design my study so that I can account  
9 for in the study design as many potential  
10 confounding agents as I can.

11 Q. Uh-huh.

12 A. Now, what is a potential  
13 confounding agent? It is a factor that may  
14 contribute to the disease that I'm studying  
15 that is not the factor under investigation.

16 Q. Right.

17 A. Okay. Now, when I design -- I  
18 would love to be able to design the  
19 confounders out of the study. Obviously that  
15:11 20 is not possible to do that in every case. So  
21 what I must do is gather information on that  
22 confounding factor so that at the -- after I  
23 have taken all of the data, then I can come  
24 back and statistically adjust my results for  
15:11 25 the contribution of that potential

51710 1254

11:11 1 confounder. I can make that adjustment.

2 Q. Right.

3 A. Now, herein lies the problem. I  
4 cannot know today what new confounding factor  
15:11 5 will be discovered tomorrow.

6 Q. Okay.

7 A. Okay? Now, if you look at the  
8 epidemiology that's been done since 1950, you  
9 had studies done, enrollments, that is, these  
15:12 10 prospective studies, enrollments were taken in  
11 the '50s and results were published in the  
12 '60s and '70s and so forth. But what has  
13 happened is that during that period of time,  
14 other confounders, that is, other risk factors  
15:12 15 for lung cancer have emerged.

16 Q. That weren't taken into account in  
17 that term?

18 A. In the early enrollments. So you  
19 have seen some of that, plus the fact if you  
15:12 20 look at the epidemiology, say, between a  
21 country like Japan and a country like the  
22 United States or the United Kingdom, you have  
23 a lower relative risk for smoking and lung  
24 cancer in Japan than you do in the United  
15:12 25 States or the United Kingdom.

51710 1255

15:12 1 Q. Okay. Well --

2 A. Now --

3 Q. -- let me take one of those at a  
4 time. Let me keep it --

15:12 5 A. I'm sorry.

6 Q. What are the additional factors  
7 that are now, at least the parent or suspect  
8 that you're specifically referring to that  
9 were not taken into account in some of the  
15:13 10 previous studies?

11 A. Some of the occupational  
12 exposures, for example, we now believe that  
13 there are certain types of chemicals that you  
14 can be exposed to of an occupational nature  
15:13 15 that have been demonstrated to be risk factors  
16 for lung cancer. There are disease factors  
17 which we now believe to be risk factors for  
18 lung cancer. For instance, it has been  
19 published that exposure to tuberculosis is a  
15:13 20 risk factor for lung cancer.

21 And in the late 1980s and going on  
22 up into the '90s -- into the -- excuse me,  
23 1980s into the 1990s, you're seeing nutrition  
24 emerge as a -- as a risk factor for lung  
15:14 25 cancer, certain nutritional factors. A report

51710 1256

15:14 1 came out of Missouri that a high fat  
2 content --

3 Q. Iowa. Isn't it Iowa?

4 A. The one I'm thinking about is a  
15:14 5 Branson study in Missouri.

6 Q. Isn't there an Iowa women's study  
7 as well?

8 A. I think there is. But now, I have  
9 not seen the --

15:14 10 Q. I didn't want to get slighted  
11 since I'm from Iowa.

12 A. Listen, I feel for you.

13 Q. I'm sorry.

14 A. This study that came from  
15:14 15 Missouri, I think it was a Branson study,  
16 showed that a high -- and this is in women,  
17 that's right, it was a women's study. But a  
18 high saturated fat content in the diet  
19 resulted in a relative risk of around six for  
15:14 20 lung cancer. You have seen studies, for  
21 example, that come out of China that -- where  
22 you have enormous lung cancer rates where less  
23 than one-tenth of a percent of the women in  
24 this study actually smoke.

15:15 25 I don't know what risk factors are

51710 1257

15:15 1 responsible for that rate. I don't know if  
2 it's genetic or not.

3 Q. Uh-huh.

4 A. But we have seen this type of  
15:15 5 thing in epidemiology. So reliance upon  
6 epidemiology to prove causation is not -- not  
7 something I would do.

8 Q. Epidemiology, though, in effect,  
9 hypothesizes an answer and then tests it?

15:15 10 A. It -- it provides you with a  
11 hypothesis --

12 Q. Uh-huh.

13 A. -- which you can then test in an  
14 animal study.

15:15 15 Q. Right. And the hypothesis that  
16 smoking causes lung cancer has been with us  
17 since -- clearly since 1950 with Sir Richard  
18 Dahl and Wynder and others. And it has been  
19 tested in various ways through epidemiology  
15:16 20 over and over and over again through decades,  
21 correct?

22 A. There have been a number of  
23 epidemiology studies for the last 30 or  
24 40 years, that's true.

15:16 25 Q. Right. And -- and rather than

51710 1258

:16 1 starting to cast doubt upon that association,  
2 the strength of the association, the -- the  
3 studies have consistently shown not only a  
4 correlation, but a very strong correlation  
15:16 5 over those years, correct?

6 A. They -- the strength of the  
7 evidence has been high, yes, that's true. .

8 Q. And in fact, the -- the most  
9 recent data that has been gathered, the -- the  
15:16 10 CPS2 data shows a varied market increase in  
11 the relative risk in many categories. Are you  
12 familiar with CPS2?

13 A. I have seen CPS2. I have seen a  
14 relative risk as high as 22, I believe, from  
15:17 15 CPS2.

16 Q. And so the hypothesis of -- let's  
17 use Dahl's hypothesis in 1950 -- has been  
18 tested in a number of different ways with  
19 different types of studies, different types of  
15:17 20 populations. And the hypothesis has held up  
21 consistently from study to study?

22 A. The hypothesis hasn't been  
23 tested. It's been reconfirmed.

24 Q. Okay.

15:17 25 A. I mean, if you do the same sort of

51710 1259

15:17 1 thing, you're going to get the same sort of  
2 result. ---

3 Q. Sure.

4 A. And what I'm saying is that  
15:17 5 epidemiology provides you with something --  
6 with a hypothesis, a theory, or whatever you'd  
7 like to call it, that you test in another  
8 arena, in another -- using a different  
9 approach, a different -- a more scientific  
15:18 10 approach.

11 Q. Uh-huh.

12 A. I can find an association. I can  
13 see the association. This begs the question,  
14 is this association causal? Now, how do I  
15:18 15 test that? Well, the only way I can really  
16 test that is to go into an animal study where  
17 I can virtually eliminate other potential  
18 confounders.

19 Q. We've mentioned occupational  
15:18 20 exposures, nutrition, and kind of the  
21 confusing data from Japan and China.

22 A. Uh-huh.

23 Q. Are there other specific instances  
24 that you --

15:18 25 A. Well, there are other things that

51710 1260

18 1 you can eliminate in an animal study. You can  
2 eliminate exposure, for example, to air  
3 pollution. You can eliminate exposure to  
4 pesticides. A very important thing that you  
15:19 5 can do with an animal study that is virtually  
6 impossible to do with a human study is that  
7 you can give an animal, a laboratory rat or  
8 rodent a virtually perfect nutrition.

9 We probably know more about the  
15:19 10 nutritional requirements of a white rat than  
11 any other living creature. We can give them a  
12 perfect fat protein carbohydrate along with  
13 minerals, vitamins, and so forth.

14 So you can eliminate that  
15:19 15 potential nutritional variable --

16 Q. Okay.

17 A. -- in animals.

18 Q. As to the kind of the specific  
19 things we've just mentioned, and we haven't  
15:19 20 discussed at length, the occasional exposure,  
21 the nutrition, and you just mentioned air  
22 pollution as a third specific area --

23 A. Uh-huh.

24 Q. -- you included among the articles  
15:20 25 that -- that were provided to me, articles

51710 1261

15:20 1 that touch on those issues. And without -- I  
2 don't really want to go through them all, but  
3 basically, have you provided, to the extent  
4 you are relying upon specific studies or  
15:20 5 specific data to make the statement that these  
6 factors are now known and relate to the issue  
7 of lung cancer, have you provided those  
8 studies to your lawyer?

9 A. I'm not sure I understand the  
15:20 10 question, but I certainly have -- one of the  
11 things that I -- one of the things that I do  
12 in my job is collect these publications.

13 Q. Uh-huh.

14 A. We have a -- a pretty extensive  
15:20 15 holding in these publications and we look  
16 for -- try to examine for new publications  
17 when they come out. So I try to make those  
18 available to management and other people  
19 and --

15:21 20 Q. Okay.

21 A. -- lawyers, too.

22 Q. Well, and the reason I asked the  
23 question the way I did is that one of the  
24 things that I've got to do today is try to  
15:21 25 figure out when you say, Mr. Thompson, one of

51710 1262

15:21 1 the problems I have with the epidemiology is  
2 it doesn't take into account air pollution,  
3 what is it that you are -- you know, what is  
4 it that you base that opinion on? What is it  
15:21 5 that you're pointing to that says air  
6 pollution has anything to do with smoking and  
7 health?

8 A. Oh, I'm sorry. It has to do with  
9 lung cancer.

15:21 10 Q. Okay. Lung cancer.

11 A. The German government has  
12 announced an intention to label diesel  
13 exhaust, for example, as a human carcinogen.  
14 The State of California Environmental  
15:21 15 Protection Agency has just released a document  
16 in which they compute a certain number of  
17 cancers per million people in California  
18 resulting from diesel exhaust.

19 The United States EPA is  
15:22 20 reassessing its particulate matter standard in  
21 the United States. I don't know where that's  
22 going to wind up. But, I mean, a lot of  
23 people are looking at that -- at that  
24 particulate matter standard to see if it  
15:22 25 protects against lung cancer, for example.

51710 1263

15:22 1 Q. Okay.

2 A. So there are halogenated  
3 hydrocarbons that appear in the atmosphere of  
4 certain -- of certain cities. Methylene  
15:22 5 chloride, for example, has been labeled as a  
6 human carcinogen.

7 So air pollution is a -- is a  
8 factor that is of concern, I mean, as far as  
9 lung cancer is concerned. Now, do I believe  
15:23 10 that it's causing a lot of human lung cancer?  
11 I don't know. I don't know how much it does.  
12 I wouldn't know how to make that computation.  
13 I'm simply saying it is a factor which has  
14 been identified. It is of concern. Where are  
15:23 15 we going to go with it? I don't know.

16 Q. Okay. The second stream of  
17 analysis is toxicology. In your opinion, does  
18 the evidence that is available today in the  
19 toxicology field support the relationship  
15:23 20 between smoking and human disease?

21 A. It does not. I have looked at the  
22 inhalation studies of cigarette smoke in  
23 laboratory animals all the way up to  
24 primates. Dr. Henry McGill at the University  
15:24 25 of Texas Southwest Medical Center San Antonio

51710 1264

15:24 1 has actually used baboons, had a colony of  
2 baboons that was smoking.

3 Q. Uh-huh.

4 A. He was looking for cardiovascular  
15:24 5 disease. Didn't find it. Studies that have  
6 been conducted, laboratory studies in rodents,  
7 rats and mice, have not shown cigarette smoke  
8 to generate lung cancer.

9 There are a number of questions  
15:24 10 about that. I mean, at one time there was the  
11 argument, well, maybe they just don't get lung  
12 cancer. But then other things would produce  
13 lung cancer in these, bischloromethylether is  
14 one that would -- diesel exhaust has been  
15:24 15 shown to produce lung cancer. So it's not  
16 that we don't have an animal that won't get  
17 lung cancer. We know that we have an animal  
18 that will get lung cancer. The argument has  
19 been made that they didn't get a high enough  
15:25 20 dose. Well, they got the maximum tolerated  
21 dose. This is the way you test --

22 Q. Uh-huh.

23 A. -- things. You -- I think it's  
24 not a good concept, but maximum tolerated dose  
15:25 25 has been -- is you push these animals to

51710 1265

15:25 1 exposure up to acute toxicity, and for the  
2 lifetime of the animal. And it didn't produce  
3 an excess of lung cancer.

15:25 4 Q. Of course, in that -- I guess in  
5 that test model, the thing that would clearly  
6 create a problem is if the -- the level of  
7 tolerance, the acute toxicity threshold for  
8 the animal, whether it be a mouse or a rabbit  
9 or a baboon, if that were lower in relation to  
15:26 10 human --

11 A. It was lower than the dose  
12 required to produce cancer?

13 Q. Right.

14 A. Well, think about this. And this  
15:26 15 is something I have to reason through and  
16 others have reasoned through it, too.

17 Q. Uh-huh.

18 A. What is the limit when you're  
19 exposing a rat to cigarette smoke? What  
15:26 20 limits your ability to get a dosing? Most  
21 agree that it's carbon monoxide. Carbon  
22 monoxide is acutely toxic.

23 Q. Uh-huh.

24 A. It binds with hemoglobin. It  
15:26 25 binds with hemoglobin and produces

51710 1266

:26 1 carboxyhemoglobin. Now, when the amount of  
2 hemoglobin in the blood exists as -- as  
3 carboxyhemoglobin exceeds 60 percent of the  
4 hemoglobin, when you have more than 60 percent  
15:27 5 the hemoglobin tied up as carboxyhemoglobin --

6 Q. Uh-huh.

7 A. -- the animal is going to die. \*

8 Q. To die?

9 A. Okay. The same is true of a human  
15:27 10 being. You hit the 60 percent mark, and you  
11 are going to die.

12 Q. Uh-huh.

13 A. Okay. Now, we push those animals  
14 all the way to 60 percent. Others have pushed  
15:27 15 them all the way up to 60 percent. What is  
16 the carboxyhemoglobin level in a smoker,  
17 two-pack-a-day smoker?

18 Q. I don't know. Do you?

19 A. Run between 6 and 10 percent.

15:27 20 Q. Okay.

21 A. So we've pushed the animal to a  
22 toxic limit. The human does not approach that  
23 toxic limit.

24 Q. Uh-huh.

15:27 25 A. So on a relative basis, you're

51710 1267

15:27 1 giving the animal a lot more cigarette smoke  
2 than you're giving a human being.

3 Q. Or at least more carbon monoxide?

4 A. More carbon monoxide, assuming  
15:28 5 that the tar and the carbon monoxide follow  
6 each other, then you've given them --

7 Q. Right.

8 A. -- a higher dose. Remember, their  
9 lungs are also considerably smaller.

10 Q. Right.

11 A. A rat lung may weigh a gram.

12 Q. And you've got -- and it seems to  
13 me, and again, I'm out of my area of expertise  
14 as well, but you've got issues that relate to  
15:28 15 breathing rate, diffusing capacity, since  
16 we're talking about a gas and solubility  
17 issues, that all these things could relate to  
18 the dose of carbon monoxide in the blood, but  
19 not have a linear relationship between the tar  
15:28 20 or the condensate dose on the surface of the  
21 tissue?

22 A. I'm not sure I follow your  
23 argument.

24 Q. Okay. Well, if we look at  
15:28 25 different -- we look at two different

51710 1268

:28 1 substances. We talked about before that in  
2 the principles of toxicology that the dose is  
3 the poison.

4 A. Sure.

15:29 5 Q. And we have to look at each  
6 substance individually, I mean, you have to  
7 look at each poison to see what -- what dose  
8 and what the dosing -- the relationship  
9 between the dose and the -- the toxicity  
15:29 10 levels are.

11 Tar, if we just use tar for now,  
12 is a different substance than the carbon  
13 monoxide?

14 A. That's true.

15:29 15 Q. And unless -- is there data that  
16 suggests that there is a straight line  
17 correlation between the -- the carbon monoxide  
18 that diffuses into the bloodstream and the  
19 amount of tar that is received into the --  
15:29 20 actually into the lung?

21 A. I'm not sure that it's a linear  
22 relationship, but there is a quantitative  
23 relationship.

24 Q. Uh-huh.

15:29 25 A. Okay. If you -- as the amount of

51710 1269

15:30 1 carbon monoxide goes up, the amount of tar  
2 also goes up.

3 Q. In the smoke?

4 A. In the smoke and in the smoker.

15:30 5 Q. Okay. Where is tar measured?

6 A. I'm sorry?

7 Q. Where do you measure tar in the  
8 smoke?

9 A. Generally you look for -- nicotine  
15:30 10 is not a good marker for tar. But you can  
11 look for other molecules that might be in the  
12 particulate fraction. Not an easy task to  
13 do. It's one that I tried to develop many  
14 years ago, and it's not an easy thing to do.  
15:30 15 But there are markers in smoke in the  
16 particulate fraction that can be measured in  
17 the blood.

18 Q. And those are the markers you  
19 tried to develop a method for labeling back in  
15:30 20 '68, '69, and '70?

21 A. Well, analytical techniques  
22 weren't very good back then as compared to  
23 today.

24 Q. Right.

15:30 25 A. And what I was trying to do -- and

51710 1270

15:31 1 you've obviously read the record -- was to put  
2 something in the tobacco that would transfer  
3 in the smoke in the particulate phase that I  
4 could then use a sensitive technique for  
15:31 5 measuring if I looked in either the blood or  
6 the urine of the animal.

7 Q. Uh-huh. Which is the way to -- to  
8 solve the problem you and I have just talked  
9 about, to determine the dose of the substance  
15:31 10 you're really thinking about, which is the  
11 tar?

12 A. Yeah. I can get at nicotine  
13 pretty well. I can get at carbon monoxide  
14 pretty well. Little tougher to get to tar,  
15:31 15 that's correct.

16 Q. And you were, in fact, involved in  
17 a project to try to do that when the program  
18 was shut down in 1970?

19 A. That was part of the -- it was  
15:31 20 part of a process in order to qualify -- see  
21 if we could get some characteristics on a  
22 smoking machine that we had developed, that's  
23 true.

24 Q. And that would be a critical piece  
15:32 25 of information in solving the puzzle that you

51710 1271

15:32 1 and I have just talked through, which is  
2 whether the dosing -- in dosing an animal to  
3 acute toxicity for carbon monoxide, in fact,  
4 is getting a dose of -- of the -- of the  
15:32 5 substance of interest, which is tar or the --  
6 or the -- the other word we used, the other  
7 terminology was the -- what was it,  
8 incomplete --

9 A. Products of incomplete combustion  
15:32 10 of tobacco.

11 Q. Thank you -- to find out the  
12 answer?

13 A. Well, you know, you can use  
14 salinasol now as a -- as a marker that can be  
15:32 15 used. It's a large molecule that comes  
16 through and it can be used now. And  
17 dotricontain is another one that comes as a  
18 very large molecule. It comes through in the  
19 particulate phase.

15:32 20 Q. When did that develop?

21 A. I'm sorry. I don't recall the  
22 exact date.

23 Q. Uh-huh. Is RJ using those markers  
24 to try to develop a dose model that allows  
15:33 25 them to determine the dose in these inhalation

51710 1272

15:33 1 studies?

2 A. I don't know that we are doing  
3 that at the moment. Some work was under way  
4 at one time at Oak Ridge, as I recall, to try  
15:33 5 to utilize these markers.

6 Q. Okay. Is there any disease, human  
7 disease, which -- for which, in your opinion,  
8 there exists sufficient animal data to connect  
9 it to smoking?

15:33 10 A. Any animal data which -- now, wait  
11 just a second. You use a -- you've connected  
12 it to smoking?

13 Q. Right.

14 A. Is there any --

15:34 15 Q. We've been talking in general and  
16 I want to just make sure -- I want to ask you  
17 the question, is there any disease in talking  
18 about the relationship between smoking and  
19 disease, for which you believe there is a  
15:34 20 sufficient acceptable animal model?

21 A. For which there is evidence in an  
22 animal that establishes causation in a human?

23 Q. Correct.

24 A. No.

15:34 25 Q. Okay.

51710 1273

15:34 1 MS. FORBES: Jeff, if this  
2 would be a good point for a leg stretch, that  
3 would be great.

4 MR. THOMPSON: Yeah, that  
15:34 5 would be fine.

6 THE VIDEOGRAPHER: Off the  
7 video record.

8 (A recess was taken.)

9 THE VIDEOGRAPHER: We're on  
15:45 10 the video record.

11 Q. (By Mr. Thompson) We're back. We  
12 have talked about epidemiology, toxicology,  
13 and if I understood your last answer  
14 correctly, it is your opinion that there is no  
15:45 15 human disease for which there is sufficient  
16 animal data to establish causation in terms of  
17 the smoking?

18 A. That's correct.

19 Q. Number 3 is chemistry. What do  
15:45 20 you mean by that?

21 A. When you're -- when you're looking  
22 at a mixture or a -- combinations of chemicals  
23 or -- the chemistry determines what -- what  
24 level of exposure we're talking about, how  
15:46 25 much is there. And what kind of reactivity

51710 1274

15:46 1 does -- do these chemicals have, how potent  
2 are they.

3 Q. The question of whether or not the  
4 chemical can cause this kind of --

15:46 5 A. It is -- is it there in a high  
6 enough concentration to be -- to be relevant,  
7 what types of chemicals are there.

8 Q. Okay.

9 A. Speciation is one thing;  
15:46 10 quantitation is another side.

11 Q. What is speciation?

12 A. Well, you -- in a mixture you  
13 have -- could have any number of chemicals.  
14 What you want to know is what species do you  
15 have.

16 Q. What type of chemical, what family  
17 do they fall in?

18 A. What families of chemicals do you  
19 have.

15:47 20 Q. In your opinion, does the  
21 information that is available regarding the  
22 constituents of smoke, and specifically the  
23 tar that we've been talking about in generic  
24 terms, satisfy this criteria? Is it the right  
15:47 25 type of chemical?

51710 1275

15:47 1 A. I don't exactly understand your  
2 question, but I can answer it this way. There  
3 are a -- a number of chemicals have been  
4 reported in cigarette smoke which have been  
15:47 5 shown to produce cancer in animals at one  
6 level -- one animal or another at one level or  
7 another.

8 Q. Okay.

9 A. So there are, in fact, known  
15:48 10 carcinogens in cigarette smoke, that's true.

11 Q. Okay. How is that different?  
12 What you've just described, the fact that  
13 there are constituents in the smoke that are  
14 known carcinogens and which show to be  
15:48 15 carcinogens in animal testing, how does that  
16 differ from the Category Number 2 that you and  
17 I just discussed? Why is that not sufficient  
18 to satisfy your animal testing requirement?

19 A. Look at Number 4.

15:48 20 Q. Okay.

21 A. And that no constituent of smoke  
22 as found in smoke has been found to cause such  
23 diseases, as found in smoke.

24 Q. Okay.

15:48 25 A. Now, they have been reported to be

51710 1276

15:48 1 in cigarette smoke or in tobacco smoke in  
2 general. But when that smoke containing that  
3 chemical is presented to the animal, that  
4 animal has not contracted the disease under  
15:49 5 investigation.

6 Q. Okay. Is that -- are you saying  
7 that that proves a negative?

8 A. No. I'm simply saying that we are  
9 still left with a moot issue. It doesn't  
15:49 10 prove that cigarette smoke will not cause  
11 cancer. But it does not support the  
12 hypothesis that it does cause cancer.

13 Q. Even though where we have isolated  
14 some component of smoke and it has been shown  
15:49 15 to cause cancer, that is not sufficient  
16 because there is data that suggests whole  
17 smoke or tar does not cause cancer?

18 A. Exact -- let me give you an  
19 example of this. A few years ago Dr. Aims was  
15:50 20 on a TV program, the Cancer Scare or something  
21 like this.

22 Q. Uh-huh.

23 A. And he was making this point. He  
24 was in a supermarket. And he was talking  
15:50 25 about carcinogens. And he picked up a -- I

51710 1277

15:50 1 think he picked up a mushroom. It was  
2 something off a supermarket counter. He said,  
3 now, you see -- he was talking to John  
4 Stossel.

15:50 5 Q. There's carcinogens in this?

6 A. There's a certain number of  
7 carcinogens in here. Am I afraid to eat  
8 this? No.

9 Now, you're correct, that is what  
15:50 10 I'm talking about. Okay. If I isolate one of  
11 those chemicals and I present it to an animal  
12 at a high enough dose -- some animal at a high  
13 enough dose, I may be able to produce cancer  
14 in that animal. But as it appears in that  
15:50 15 mushroom, it doesn't -- doesn't cause cancer.

16 Q. Because of the quantification  
17 component of this question or the speciation?

18 A. I don't know. I don't know.  
19 That's a good -- that's -- that's a good  
15:51 20 question. I don't know the exact answer to  
21 that. Is it because something else in the  
22 mushroom blocks the action of that  
23 carcinogen?

24 It's an interesting sort of  
15:51 25 argument. I know a few years ago -- it's been

51710 1278

15:51 1 a number of years ago now -- there was the  
2 argument over whether or not phenols were  
3 co-carcinogens or promoters, phenolic  
4 compounds.

15:51 5 Q. Uh-huh.

6 A. And Dr. Van Duren at New York  
7 University did a skin painting study, which  
8 is, once again -- but he used the promoter  
9 or -- excuse me. The alleged carcinogen was  
15:52 10 benzopyrene, which is a polycyclic aromatic  
11 chemical, and he used the phenols. And it  
12 turned out that the phenols, phenolic  
13 compounds in his test blocked the action of  
14 benzopyrene.

15:52 15 Q. Uh-huh.

16 A. Now, I've heard all sorts of  
17 arguments about this phenolic compounds in  
18 general are antioxidant-type compounds. They  
19 are -- they act to scavenge free radicals, and  
15:52 20 as such, sort of block --

21 Q. Block the activity?

22 A. -- oxidative activity. Now, does  
23 that mean that the benzopyrene, when it acts  
24 as a carcinogen, acts as an oxidizing agent?

15:52 25 I don't think so. I don't know what it was

51710 1279

15:52 1 doing. I'm just simply saying it's not always  
2 easy to interpret the results you get from any  
3 study. And that's one that's still sort of  
4 confusing. I mean, I am --

15:52 5 Q. But in this regard, you agree that  
6 there is animal data out there that shows  
7 that -- at least constituent parts of smoke  
8 are carcinogens?

9 A. When tested in one animal species  
15:53 10 or another at a high enough dose, they produce  
11 cancer in animals, that's correct.

12 Q. Your objection, however, is that  
13 when tested either as a component of tar or  
14 whole smoke or at levels that would occur in  
15:53 15 tar or whole smoke, it does not create cancer  
16 in an animal model?

17 A. That -- the problem is as it  
18 appears in smoke -- the problem is -- and  
19 that's true. I mean, when cigarette smoke  
15:53 20 containing some of these chemicals is  
21 presented to animals by inhalation, it has not  
22 produced lung cancer in these animals.

23 The issue, I think, that you're  
24 asking in the question was, is it dosimetry or  
15:54 25 something like that. I don't know whether

51710 1280

:54 1 it's -- whether it's dose or whether something  
2 in the smoke is protective.

3 Q. Okay. Are there -- is there no  
4 animal testing that has created cancer in  
15:54 5 inhalation studies with smoke?

6 A. No animal study. There have been  
7 studies in the 1970s in hamsters --

8 Q. Uh-huh.

9 A. -- in which laryngeal cancer was  
15:54 10 observed in a set of hamsters -- two sets of  
11 hamsters. One very small set was exposed by  
12 Dr. Humberger.

13 Q. Syrian hamsters, in fact?

14 A. Syrian golden hamsters, bio 1516  
15:55 15 was his -- it was an inbred string of hamsters  
16 that he created -- that ostensibly he created  
17 to be particularly susceptible to cancer.

18 Now, I have looked at that study  
19 and talked to other people about the study.  
15:55 20 An attempt was made to repeat the study in  
21 hamsters.

22 Q. In Germany?

23 A. No, that was a Donwille study.  
24 That was a Donwille study that was also done  
25:55 25 in the '70s. And I'm not sure what the date

51710 1281

15:55 1 was on that study. Now, that was a much  
2 bigger study than Humberger's study.

3 Q. Uh-huh.

4 A. The Humberger study was done in  
15:55 5 this rather uniquely bred-up species of  
6 animals. And they observed or reported an  
7 excess of laryngeal cancer in these animals.

8 An attempt was made to reproduce  
9 that study at Betel Northwest. In other  
15:56 10 words, Dr. L. Weiner did a -- a similar study,  
11 not -- but he didn't use the bio 1516. He  
12 told me he tried to get the bio 1516, but he  
13 couldn't -- he never was able to obtain any of  
14 them. And I don't even know if that string  
15:56 15 still exists, that bio 1516.

16 His problem was he didn't -- he  
17 didn't find the same result that Dr. Humberger  
18 found. His problem was that the animals that  
19 were exposed to cigarette smoke outlived the  
15:56 20 animals that were not exposed to cigarette  
21 smoke, had a longer life. I don't know why.  
22 He didn't try to explain that. He just  
23 reported that.

24 Now, the other study that you're  
15:56 25 referring to is the Donwille study, very large

51710 1282

15:56 1 study. My recollection of that study is that  
2 he started off with maybe four to 6,000  
3 animals. It was an enormous number of  
4 hamsters that he used in that study. And he  
15:57 5 used a number of different types of  
6 cigarettes, experimental cigarettes as well  
7 as, I think, some standard cigarettes, too.\*

8 Now, he got into the study about  
9 four to six weeks into the study and  
15:57 10 discovered that those animals were infected.  
11 His entire colony had something. I -- I would  
12 have to go back and look at the study.

13 Q. Uh-huh.

14 A. He treated them -- now, today, if  
15:57 15 something like that occurred today, the entire  
16 set of animals would be euthanized, destroyed,  
17 you start over.

18 Q. Uh-huh.

19 A. Because you don't know how this  
15:58 20 disease compromises your animal, how it may  
21 sensitize your animal. But he did something  
22 else. He treated the animals with some drug  
23 that's used to treat this particular type of  
24 parasite.

15:58 25 Q. Uh-huh.

51710 1283

15:58 1 A. So -- okay, now, that's the first  
2 time. So he goes on, he continues his study,  
3 which violates good laboratory practice --  
4 today's good laboratory practice. Then he got  
15:58 5 into the study a little bit longer. And once  
6 again, I'd have to go back and look, it may  
7 have been six months into the study, and they  
8 were -- he found them infected with something  
9 else. And he treated them again with some  
15:58 10 other drug.

11 So he's -- now he's had two  
12 different infections, two different drugs in a  
13 set of animals that should have been  
14 destroyed.

15:58 15 So the results of that study are  
16 just of no value to me whatever. When I  
17 couple that evidence with the fact that no one  
18 has ever reported an excess of laryngeal  
19 cancer in other laboratory animals exposed to  
15:59 20 cigarette smoke, it makes that very suspect.

21 In other words, I don't have a  
22 reproducible study. It has not been  
23 reproduced in more than one string of animal.  
24 And I've got one set of animals, even though  
15:59 25 it was a large study, potentially would have

51710 1284

15:59 1 been a very good study to have, but they were  
2 infected. And they were treated with -- with  
3 the set of drugs that -- I don't know how that  
4 affected the outcome of the study.

15:59 5 Q. Uh-huh.

6 A. So now, those are some studies --  
7 two studies. That is, a study by Humberger\*  
8 and a study by Donwille that have been  
9 reported to show an excess of laryngeal  
16:00 10 cancer -- laryngeal cancer in animals exposed  
11 to cigarette smoke.

12 Q. In Paragraph 1, you make the  
13 statement that -- that causation, making the  
14 determination of causation is a matter of  
16:00 15 judgment based on an analysis of the things  
16 we've just kind of gone through, correct?

17 A. Well, I quoted the '64 surgeon  
18 general's report. Is that the one you're  
19 looking at?

16:00 20 Q. Uh-huh. Do you agree with that,  
21 that that's the appropriate --

22 A. I agree that that's what they  
23 said, that their conclusion was a matter of  
24 judgment, and that no one on that committee  
16:00 25 used causation in an absolute sense or some --

51710 1285

16:00 1 words to that effect. I don't remember the  
2 exact quote.

3 Q. Uh-huh.

4 A. But they did use that as a matter  
16:00 5 of judgment.

6 Q. Okay. And in the context of a  
7 committee charged with making a public health  
8 recommendation, do you disagree with their  
9 judgment?

16:05 10 A. Having not been present at the  
11 discussions, knowing what the context was,  
12 what they were doing, I don't know how to  
13 comment on that. That was a matter of their  
14 judgment.

16:05 15 Q. Uh-huh.

16 A. I don't think that the evidence I  
17 have seen would support that conclusion in a  
18 scientific sense. They are making -- making a  
19 judge -- a judgment from a public health  
16:05 20 official's consideration.

21 Q. Uh-huh.

22 A. And so I don't know how to answer  
23 the question.

24 Q. Would you agree, though, that --  
16:05 25 that those are really two different inquiries,

51710 1286

16:05 1 that on one hand we've talked about the  
2 scientific inquiry and scientific certainty,  
3 which is what you and I have been talking  
4 about for some time today, and the surgeon  
16:05 5 general's report was the conclusions were  
6 drafted in the context of making a judgment in  
7 terms of, in effect, public policy as it  
8 related to health?

9 A. There are two different exercises,  
16:05 10 I think that's correct.

11 Q. Right. And do you -- I mean, do  
12 you disagree with the conclusions that they  
13 have reached and the -- what they have focused  
14 on, if you could put your -- take your  
16:05 15 absolute scientist hat and put on your public  
16 policy hat for a second? I mean, is it good  
17 public policy to err on the side of caution  
18 when it comes to issues about cancer and  
19 chronic disease?

16:05 20 A. I don't have a public health  
21 official's hat. The way that this has played  
22 out is that Congress has acted on public  
23 health official's findings, promulgated  
24 warnings for cigarettes, and our company  
16:05 25 abides by that law. We put them on there.

51710 1287

16:05 1 Q. Uh-huh.

2 A. I'm not a warnings expert. I  
3 don't try to set up criteria by which one  
4 establishes some sort of warning. It's not an  
16:05 5 exercise that I -- that I go through. That's  
6 the best way I know to answer your question.

7 Q. In looking at -- since you don't  
8 have a public policy hat, and I will accept  
9 that, but looking at it from the outside and  
16:05 10 looking at the decisions -- the data that they  
11 have reviewed over time and the  
12 recommendations that have been made, do you --  
13 is it your opinion that they are wrong -- that  
14 they have made the wrong judgment in a public  
16:05 15 policy sense?

16 A. Well, once again, that's a context  
17 that I don't -- I don't know how to deal with  
18 that. From a purely scientific standpoint, I  
19 don't agree with the conclusion, and I don't  
16:05 20 have a public policy or a public health  
21 official hat.

22 Q. Flipping through here. It looks  
23 like we've covered Number 2. We've talked  
24 about the weaknesses and the comparison to  
16:05 25 controlled laboratory animal experiments. Is

51710 1288

16:05 1 there anything we just have not even discussed  
2 that relates to your opinions in terms of the  
3 epidemiology and the -- the absence of an  
4 animal model?

16:05 5 A. I'm sorry. I don't understand the  
6 question.

7 Q. Is there a topic in a general  
8 sense that we just have not talked about that  
9 would fall under your Paragraph Number 2? I  
16:06 10 will withdraw the question. That's fine.

11 In Number 6, Number 6 states that  
12 you're expected to testify that diseases  
13 associated with smoking in humans occur in  
14 both smokers and nonsmokers. In your opinion,  
16:06 15 does that exclude smoking as a cause?

16 A. No, it does not. I mean, smoking  
17 may be a cause. It doesn't exclude it, no.

18 Q. Okay.

19 MR. THOMPSON: Why don't we  
16:07 20 take a short break and let me sort through  
21 some stuff.

22 THE VIDEOGRAPHER: Off the  
23 video record.

24 (A recess was taken.)

16:07 25 THE VIDEOGRAPHER: We're on

51710 1289

16:13 1 the video record.

2 Q. (By Mr. Thompson) Dr. Simmons,  
3 your report also refers to --

4 A. I'm sorry?

16:13 5 Q. If you look at Paragraph 7, 8, and  
6 9 -- deal with your work at the biological  
7 research division back in 1968 through 1970.

8 A. That's correct, yes, sir.

9 Q. You've given testimony about it  
16:13 10 before. I've read it. I don't intend to go  
11 through all the excruciating details again,  
12 but suffice it to say you were working on the  
13 project that you and I mentioned, which is the  
14 tagging project?

16:13 15 A. That was -- that was one -- one  
16 part of it, yes.

17 Q. What other specific projects were  
18 you working on?

19 A. The -- okay. There were two  
16:14 20 projects that -- two other -- well, gee. The  
21 tagging project that you're -- that you were  
22 referring to, that is the labeling, how can I  
23 label cigarette tar with a specific compound  
24 that will allow me to quantify the dose of tar  
16:14 25 that an animal received when exposed to smoke,

51710 1290

14 1 okay. That was the -- that's one.

2 Q. Right.

3 A. Okay. Now --

4 Q. Was there a name for that program,  
16:14 5 a specific name?

6 A. You see, that particular task was  
7 part of the task in developing a smoking  
8 machine.

9 Q. Okay.

16:14 10 A. A machine that would allow us to  
11 expose laboratory rodents to cigarette smoke.

12 Q. Okay.

13 A. So that was -- that was in support  
14 of that type of -- and keep in mind, back in  
16:14 15 those days, we could not even measure nicotine  
16 in the urine of the exposed animals. That was  
17 a very difficult thing to do in those days.  
18 You follow what I'm talking about?

19 Q. Uh-huh.

16:15 20 A. Today that's routine. I mean,  
21 that's a routine thing, easy to do. We can  
22 measure nicotine, cotinine -- what am I  
23 saying? We can measure 10 metabolites  
24 probably in the urine of exposed animals. In  
16:15 25 those days it was very difficult to even do

51710 1291

16:15 1 that, to measure nicotine. We couldn't do  
2 it.

3 All right. Now, so that was the  
4 smoking machine project. The most interesting  
16:15 5 project that I was working on was the project  
6 that dealt with the pharmacological  
7 endeavor --

8 Q. Uh-huh.

9 A. -- that is to produce for  
16:15 10 Pennekin 4 --

11 Q. No, the other one.

12 A. The other one, that's right.

13 Q. I'm sorry.

14 A. The most interesting thing out of  
16:15 15 that was the fact that a chemical had been  
16 synthesized that we referred to as a  
17 cyclomethicone, which had the ability to lower  
18 cholesterol in laboratory animals. It was not  
19 a trivial effect. It was -- when compared to  
16:16 20 the compound of the drug that was on the  
21 market at that time, it was a sledge hammer  
22 effect. It had a marked effect in -- in  
23 reducing serum cholesterol in these animals.  
24 So that was a very interesting project that I  
16:16 25 was working on at that time.

51710 1292

16 1 Q. And what else?

2 A. And we had this other project, we  
3 owned a starch company called Pennekin 4.

4 Q. Cedar Rapids, Iowa.

16:16 5 A. Really? I knew it was Iowa. I  
6 didn't know where it was. I knew it was an  
7 Iowa company -- and they made, among other  
8 things, I mean, they had a lot of products,  
9 but they made a lot of products. But they  
16:16 10 made syrups that would be for the breakfast  
11 table that you would put on pancakes and  
12 waffles and things like that. And we had a  
13 project to see if we could increase the  
14 sweetness of these syrups.

16:17 15 Now, I see you look like that's a  
16 trivial thing, you just put more sugar in  
17 there, right? Well, that's not the way we  
18 were trying to do it. The idea was to -- if  
19 you look at the sweetness of the various  
16:17 20 saccharides, the various sugars, what you find  
21 is that glucose is not very sweet.

22 Q. Uh-huh.

23 A. But fructose is very, very sweet,  
24 eight to 10 times, maybe even 20 times sweeter  
17 25 than glucose. Now, there is an enzyme called

51710 1293

16:17 1 a glucose isomerase --

2 Q. Okay.

3 A. -- which will convert glucose to  
4 fructose. And the idea was to commercialize  
16:18 5 that process. Can I take this enzyme and use  
6 it to take a solution of glucose, percolate it  
7 over the enzyme, and convert the glucose to  
8 fructose, which is very, very sweet.

9 Well, I was working on -- we knew  
16:18 10 it would work. The idea was, will it work on  
11 a commercial scale. And I was trying to find  
12 a way to immobilize the enzyme on a column so  
13 that I could percolate a solution through the  
14 column, start out with glucose at the top and  
16:18 15 come out with a mixture of fructose and  
16 glucose at the bottom, which increases the  
17 sweetness.

18 I did not solve the problem, but  
19 it was solved. A patent was taken out at  
16:18 20 Reynolds and, of course, it's run out now.  
21 But that was eventually solved. But I was  
22 working on that project as well.

23 Q. Uh-huh.

24 A. So those were the three projects  
16:19 25 that I worked on during that period of time.

51710 1294

:19 1 The animal exposures --

2 Q. Smoking machine?

3 A. But that -- it was not just that.

4 Q. Uh-huh.

16:19 5 A. It was the animal exposures and  
6 development of a way or techniques that  
7 allowed -- would allow us to study the impact  
8 of cigarette smoke on certain processes in the  
9 lung. There was a material, a lipid  
10 bacteria --

11 Q. Surfactant?

12 A. -- pulmonary surfactant.

13 Q. Uh-huh.

14 A. There was an enormous literature  
16:19 15 at that time. See, this was '68 and '70, and  
16 I had found enormous literature published  
17 papers on pulmonary surfactant.

18 There was speculation -- a book  
19 had opinion written on this -- Emilio  
16:19 20 Scarpelli wrote a book on pulmonary  
21 surfactant.

22 There was speculation that  
23 emphysema -- cigarette smoke generated  
24 emphysema by some effect, some influence over  
:20 25 pulmonary surfactant.

51710 1295

16:20 1 Now, that's a very broad -- I used  
2 a very broad -- "some effect."

3 Q. Uh-huh.

4 A. Now, in order to study that or to  
16:20 5 test that, we were trying to develop  
6 laboratory techniques, analytical -- I say  
7 laboratory techniques -- some way of analyzing  
8 pulmonary surfactant, looking at enzymes  
9 involved in pulmonary surfactant, synthesis  
10 and turnover, extraction techniques that would  
11 allow us to get the pulmonary surfactant out  
12 so we could look at it.

13 Q. And you were looking at the  
14 cellular level, in fact, or trying to?

16:20 15 A. Actually subcellular.

16 Q. Molecular?

17 A. Trying to get down to the  
18 molecular level, exactly. That was -- that  
19 was the idea.

16:21 20 Q. Uh-huh.

21 A. Now, it's easy to say that, but  
22 it's quite a difficult matter to actually do  
23 it.

24 Q. Uh-huh.

16:21 25 A. But we were -- we were giving it a

51710 1296

.21 1 shot. I mean, we were trying --

2 Q. Uh-huh.

3 A. -- to develop these techniques to  
4 see if we could find a way to study this in  
16:21 5 animals exposed to cigarette smoke.

6 Q. In effect, you were looking for  
7 what we talked about before, which is the --

8 A. The molecular level.

9 Q. To show changes at the molecular  
16:21 10 level?

11 A. Go to the molecular level. That  
12 was -- that was the idea. See, this is what  
13 we were looking to get toward. But the first  
14 thing you've got to do is develop your  
16:21 15 techniques.

16 When you do a biochemical study,  
17 quite often you find you have to develop  
18 techniques -- there are no canned techniques.  
19 Aha, I can pull a technique off the shelf for  
16:21 20 studying pulmonary surfactant or pull one off  
21 the shelf for studying an enzyme.

22 The first thing you try to do is  
23 reproduce what somebody else may have done in  
24 the literature.

25 Q. Uh-huh.

51710 1297

16:22 1 A. You have his -- I've got his  
2 paper. He says -- here's his experimental  
3 design, here's his techniques, here's the way  
4 he made a measurement, can I reproduce that.

16:22 5 Q. Uh-huh.

6 A. So often that's the way you  
7 start. And then you try to improve on that.

8 Q. So you, in fact, were -- for how  
9 long did you expose the animals in -- in the  
16:22 10 preliminary phases of the study?

11 A. Okay. Now, for virtually -- for  
12 most of that work, we were not working with  
13 the animals that were exposed on the smoking  
14 machine. That was a little bit different type  
16:22 15 of experiment.

16 Q. You were trying to lay the  
17 groundwork for how you get --

18 A. Yeah. What had been done in that  
19 experiment with those laboratory rats was  
16:22 20 using labeled polymictic acid, C-14 labeled  
21 polymictic acid to see if the smoke affected  
22 the uptake of polymictic acid. This is a  
23 complicated study. Are you sure you want to  
24 hear this?

16:23 25 Q. I don't, because I have read most

51710 1298

:23 1 of it already.

2 A. Okay.

3 Q. But I guess what I would -- just  
4 to try and confirm the facts, how -- for how  
16:23 5 long were the animals on that aspect of the  
6 big program actually exposed to cigarette  
7 smoke?

8 A. I'm sorry, I don't recall. I  
9 don't recall the exact length of time they  
16:23 10 were exposed. But we were working with New  
11 Zealand whites, which you are probably aware  
12 of, which is a very large animal.

13 Q. Right.

14 A. It's a five-kilo animal. It gives  
16:23 15 you a bigger lung to work with.

16 Q. Right.

17 A. And we had a small number of those  
18 animals. I think there were maybe 13, 14 of  
19 those animals that we were working with.

16:23 20 Q. Uh-huh.

21 A. Using an exposure technique that  
22 was really pretty primitive. And I'm not sure  
23 how long those animals were exposed. I would  
24 have to go back --

:24 25 Q. Uh-huh.

51710 1299

16:24 1 A. -- and I just haven't looked at  
2 that evidence in a long time.

3 Q. Okay. You've already testified on  
4 numerous occasions that it's your  
16:24 5 understanding that the program was shut down  
6 for, I guess, program and efficiency reasons.

7 A. Business reasons.

8 Q. Business reasons. And it's your  
9 opinion or it's your understanding it had  
16:24 10 nothing to do with the work that was actually  
11 being conducted in terms of the --

12 A. I don't believe that. I don't  
13 believe that it was.

14 Q. Do you know? I mean, do you know  
16:24 15 why the management decided to shut it down?

16 A. I know what they told me, and I  
17 have no reason to disbelieve that.

18 Q. Okay.

19 A. I was not at management meetings  
16:24 20 where the decisions were made, but I know what  
21 was said to us.

22 Q. Uh-huh.

23 A. And I believe that. I mean, I  
24 just -- one of the things -- I don't know if  
16:24 25 you were aware of this part of it or not. I

51710 1300

:25 1 will give you another piece of information. I  
2 was working with -- I had made an interesting  
3 finding. Through electrophoresis I had  
4 found -- my goodness, look at all of these  
16:25 5 wonderful enzymes that I'm finding when I  
6 extract a rabbit lung. Well, I couldn't find  
7 that in the literature. I couldn't see that  
8 that was in the literature. Looked like I had  
9 made a new finding.

16:25 10 Q. Right.

11 A. So I was pretty keyed up and ready  
12 to go about this. Well, at that time our  
13 group of biochemists and so forth had a good  
14 relationship -- we still do -- with Bowman  
16:25 15 Gray -- with the biochemistry department of  
16 Bowman Gray. And when they had visiting  
17 seminar presenters or speakers come in, we  
18 would get a notice and we would get invited to  
19 come over and listen to it.

16:25 20 We got an invitation that there  
21 was a Dr. Albert Votter from the Web Wearing  
22 Institute in Denver that was going to make a  
23 presentation.

24 Q. Uh-huh.

:25 25 A. This is interesting. And he

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16:26 1 was -- it talked about lung enzymes. And I  
2 thought, you know, we ought to go hear this.  
3 So we get over there. Dr. Votter is a  
4 wonderful, very objective, very clear, very  
16:26 5 lucid scientist -- makes a good presentation.  
6 And he presented exactly what I was doing in a  
7 laboratory. In other words, he was doing that  
8 out there, he was looking at that particular  
9 set of estrolytic enzymes at the Web Wearing  
16:26 10 Institute.

11 Now, the two things that happen  
12 when you see something like that, first thing  
13 you think is, hey, I'm in the mainstream, I'm  
14 doing -- and then you think, oh, he scooped  
16:26 15 me.

16 Q. Uh-huh.

17 A. But at the -- the fact of the  
18 matter is, his ideas were very consistent with  
19 the ideas that I had.

16:26 20 A number of years later when I  
21 came back -- and I talked to him. We had a  
22 long conversation after his presentation about  
23 what I was doing, what he was doing, and what  
24 we were finding and all that sort of thing.

16:27 25 Q. Right.

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:27 1 A. And a number of years later when I  
2 came back to Reynolds, I discovered that he  
3 had -- he was funded by something called the  
4 AMA-ERF fund. I don't know if you have ever  
16:27 5 heard of that. The cigarette industry had put  
6 up something like \$10 million into a fund,  
7 research fund that was administrated by the  
8 American Medical Association.

9 Q. Uh-huh. I am familiar with that.

16:27 10 A. And they had directed some of that  
11 fund over to Dr. Votter, so the tobacco  
12 industry was paying for the same sort of work  
13 I was doing in-house over at -- over at the  
14 Web Wearing Institute.

16:27 15 Q. Uh-huh.

16 A. But there were other people. I  
17 mean, that's just an example because I got to  
18 hear Dr. Votter.

19 And later on in the '70s, he  
16:27 20 published that work and some very interesting  
21 publications came out.

22 Q. Okay. Do you -- were you aware at  
23 the time of any agreement between your company  
24 and others not to conduct in-house testing,  
25 in-house animal testing?

16:28 1 A. I heard nothing along those  
2 lines. I just don't recall anything along  
3 those lines at all.

4 Q. Okay. And since you've returned  
16:28 5 to the company, have you come to learn of any  
6 such agreement between the companies?

7 A. I don't know that any kind of  
8 agreement like that ever existed. I'm not  
9 privy to that.

16:28 10 Q. Okay. You just don't know?

11 A. I don't know.

12 Q. When it was all shut down, kind of  
13 jump to the end of the story here -- and  
14 you've testified that they collected your data  
16:28 15 and kept it and didn't give it back; is that  
16 correct?

17 A. The -- when you leave -- it's  
18 correct. They took up -- our laboratory  
19 notebooks were taken up, our reports were  
16:29 20 taken up, of course, and were placed into  
21 archives and they still exist in those  
22 archives.

23 Q. Okay.

24 A. I mean, all of that information  
16:29 25 still exists. That is not -- there's nothing

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:29 1 sinister about that.

2 Q. Okay.

3 A. I mean, when I left Borroughs  
4 Wellcome, they took all my notebooks up there,  
16:29 5 too, but there's nothing sinister about that.

6 Q. But all the data still exists?

7 A. The data exists, all -- everything  
8 that I was doing, everything that we had done,  
9 recorded in our notebooks, all of that is  
16:29 10 still there.

11 Q. Okay.

12 A. And I -- I've seen it.

13 Q. Okay. What happened to the  
14 machines?

16:29 15 A. Okay. Which machines are we  
16 talking about here, the smoking machines?

17 Q. The smoking machines.

18 A. Now, it is my understanding -- and  
19 this was contract effort that was outside of  
16:29 20 my own personal involvement -- that that

21 machine was turned over to the -- at that time  
22 it may have been called Tobacco Research  
23 Council. But I think it's since been called  
24 the Council for Tobacco Research or  
17:30 25 something. Anyhow, there was a change.

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16:30 1 Q. Right.

2 A. It's my understanding it was  
3 turned over to that organization for potential  
4 use in studies in which animals would be  
16:30 5 exposed to cigarette smoke.

6 Q. Uh-huh.

7 A. That's my understanding of it.

8 Q. Are you aware of whether or not  
9 CTR ever used the machines for that purpose?

16:30 10 A. I don't have firsthand knowledge  
11 of that. It is my understanding that they did  
12 not, that there were other machines in  
13 competition with the one that we were trying  
14 to develop which may have been superior to the  
16:30 15 ones that we developed. The fact of the  
16 matter is, in those days, animal exposure  
17 equipment was primitive.

18 Q. Uh-huh.

19 A. I mean, it was difficult to  
16:30 20 expose -- you could expose animals easily  
21 enough to a pure gas.

22 Q. Right. But the particulates  
23 created a problem?

24 A. A big problem. If the particulate  
16:31 25 condenses on the animal's fur, I don't care

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16:31 1 whether it's diesel exhaust or cigarette smoke  
2 or coal-fired gas flint --

3 Q. Right.

4 A. -- they're going to groom  
16:31 5 themselves and now you're going to have  
6 exposure --

7 Q. Ingestion?

8 A. -- from inhalation, you're going  
9 to have exposure from ingestion, and you might  
16:31 10 have some internal exposure, too. I mean,  
11 there's just too many different ways.

12 Q. Uh-huh.

13 A. So it's a complicated thing to  
14 expose to a nongaseous material.

16:31 15 Q. Because you get, in effect,  
16 condensate on the apparatus -- on the inside  
17 of the apparatus itself?

18 A. Well, think about what I just  
19 said. You've got it on the fur. Okay. They  
16:31 20 are going to ingest it. They're probably  
21 going to get it on their skin as well as  
22 inhale some of it. Now you've got about three  
23 different levels of exposure when you're  
24 shooting for inhalation.

16:32 25 Q. Three in one?

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16:32 1 A. Hey, super dose.  
2 Q. What happened to the rabbits?  
3 A. I'm not sure what happened to -- I  
4 feel like they were euthanized. It's my  
16:32 5 recollection those animals were euthanized.  
6 I'm not sure.  
7 Q. By somebody in the group before --  
8 A. Somebody in the animal holding  
9 facility --  
16:32 10 Q. Uh-huh.  
11 A. -- probably.  
12 Q. Do you have any knowledge as to  
13 whether or not there were -- there were any  
14 tissue samples collected, lungs collected, any  
16:32 15 pathology done on the carcasses?  
16 A. As animals were sacrificed, there  
17 was pathology done on the animals that had  
18 been sacrificed, but the ones right at the end  
19 of the study, when they were euthanized, I  
16:33 20 don't know if those animals were worked up or  
21 not. I don't recall.  
22 Q. But those would have been the  
23 animals who had been exposed for the longest  
24 period of time?  
16:33 25 A. Possibly, possibly. I'm not

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:33 1 sure -- if you have a serial introduction of  
2 animals into an exposure group, you might --  
3 see, I joined in December of '68. Exposures  
4 were under way then, but animals had already  
16:33 5 been exposed and sacrificed. We were working  
6 up samples all the time.

7 Q. Uh-huh.

8 A. So I'm not sure how long an  
9 exposure took place.

16:33 10 Q. Okay.

11 A. I would have -- I may be able to  
12 find that out. I might be able to look that  
13 up.

14 Q. Uh-huh. Certainly, I guess, in  
16:34 15 the context of what we've been discussing all  
16 day that the value, regardless of the  
17 limitations, of having access to these animals  
18 that have been exposed for whatever period of  
19 time, years, months, was valuable information  
16:34 20 that could have -- could have shed some light,  
21 perhaps, on some of the issues you were  
22 investigating?

23 A. If -- okay. One, if we had the  
24 techniques to look, and if we could  
:34 25 demonstrate that the animals did not have some

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16:34 1 disease which fouled everything up, which  
2 fouled up your measurements. What we've found  
3 at one time -- remember, we were looking, we  
4 were extracting a lung, we were trying to get  
16:34 5 the pulmonary surfactant out, we were looking  
6 at the fatty acids attached to the pulmonary  
7 surfactant. We even found evidence at one  
8 time that at least one of those animals may  
9 have had tuberculosis. But certainly other  
16:35 10 tests had been done which demonstrated that  
11 the animals, at best, had some sort of  
12 pneumococcal infection, which is not  
13 surprising when you consider the way the  
14 animals were exposed, which was a very  
16:35 15 primitive exposure at that time.

16 The animals really were to serve  
17 one purpose. And that is to help us to  
18 provide samples for us to develop techniques.  
19 We didn't have a large sample size. My  
16:35 20 recollection is that last group of animals was  
21 only about 12 to 14 animals. I've forgotten  
22 the exact size. This probably was not a large  
23 enough sample to give you a statistically  
24 relevant result.

16:36 25 Q. In your opinion, did any of those

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:36 1 animals develop pathologic changes that looked  
2 like emphysema?

3 A. Now, I didn't read the slides. I  
4 didn't -- I'm not a pathologist. I didn't  
16:36 5 read the slides.

6 Q. Uh-huh.

7 A. You could certainly see  
8 differences in the individual animals. There  
9 were certain biochemical differences that I  
16:36 10 was looking at, indicating changes in the  
11 pulmonary surfactant.

12 Now, does this mean that the  
13 animals had emphysema? Not to me. When you  
14 consider the fact that they -- they were  
16:36 15 infected, they did carry a -- some sort of a  
16 pneumococcal infection. We had driven  
17 infection down into their lung by the way we  
18 were exposing the animals. It's not  
19 surprising that you would find changes, but we  
16:37 20 were seeing these changes in both controlled  
21 animals, that is, the so-called sham-exposed  
22 animals as well as the smoke-exposed animals.

23 So when you have a bacterial  
24 infection, you're going to get differences and  
:37 25 changes which are -- may be related to the

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16:37 1 infection that you're dealing with.

2 We certainly didn't have a quality  
3 of data -- let me give you an idea. If we had  
4 written this study up and said, okay, here's  
16:37 5 our write-up, we've got a limited number of  
6 animals, we've got this type of evidence. And  
7 by the way, these animals -- at least some of  
8 these animals had pneumonia. That study would  
9 not be accepted for publication.

16:38 10 Q. So like the Humberger study and  
11 the German study we discussed before, even  
12 though there might have been some evidence of  
13 changes, in your opinion, not a valid animal  
14 study?

16:38 15 A. Not a valid animal study for a lot  
16 of reasons. Now, the Humberger study -- don't  
17 misunderstand me, I didn't say that the  
18 Humberger study had -- were infected animals.

19 Q. I understand. It wasn't  
16:38 20 reproduced?

21 A. It was not reproduced, but it was  
22 more than that. I'm -- he indicated he had  
23 bred up a specially sensitive group of  
24 animals. I'm not sure what -- how relevant  
16:38 25 that is to a -- to a study, to getting the

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38 1 information. And it was a relatively small  
2 study at that, but --

3 (Deposition Exhibit 6  
4 was marked.)

16:38 5 Q. Let me show you what I've marked  
6 as Exhibit 6. It's just the cover page to the  
7 Brubaker report. You've seen it before, I'm  
8 sure.

9 A. I have seen a redacted version of  
16:39 10 the Brubaker report. I have not seen an  
11 unredacted version.

12 Q. Okay. Do you know why more than  
13 10 years later somebody was asked to go back  
14 and reconstruct the events that you and I have  
16:39 15 just discussed?

16 A. I was not --

17 MS. FORBES: Wait one  
18 second. Just for the record, and I understand  
19 that this is not going to be resolved today.  
16:39 20 And this is the only objection we can make is  
21 that this is privileged and confidential,  
22 produced for Jones, Day, Reavis & Pogue, the  
23 law firm representing R.J. Reynolds. You may  
24 examine.

39 25 Q. (By Mr. Thompson) And just so

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16:39 1 it's clear, I don't think you were involved in  
2 discussions with counsel about this, and so  
3 what I'm asking you about is your own personal  
4 knowledge.

16:39 5 Do you have any knowledge  
6 developed just from your work and not from  
7 discussions with counsel about why in 1985  
8 Paul Brubaker was asked to value the issues  
9 that we just discussed?

16:40 10 A. I have not officially stated --  
11 nobody has told me why that was done.

12 Q. Do you agree with his analysis of  
13 your work and your data?

14 A. Where do we stand here?

16:40 15 MS. FORBES: Well --

16 MR. THOMPSON: Go ahead.

17 MS. FORBES: And again, under  
18 the privilege and confidential objections we  
19 may raise, and this document that is marked as  
16:40 20 Simmons 6 indicates a cover page that is being  
21 provided in accordance for a court order for  
22 in-camera inspection in the Keuper case, which  
23 is the case that was tried in Illinois, not  
24 the Texas case. I think we've got the right  
16:41 25 to maintain the privileged and confidential

1 nature of this document.

2           You may examine on what facts he  
3 knows about this document. I don't think  
4 that -- that is separate and apart for any  
16:41 5 privileged and confidential relationship, if  
6 that's clear.

7           MR. THOMPSON: And on the  
8 record I want to make sure that I am clear. I  
9 am not asking you to discuss with me any  
16:41 10 communications that you might have had with  
11 counsel, privileged communications. All I  
12 want to know -- and let me see if I can lay a  
13 foundation that will make it clear.

14           Q.     (By Mr. Thompson) You're  
16:42 15 familiar -- you've read the report?

16           A.     I've read a redacted version. And  
17 I didn't have it --

18           Q.     Right.

19           A.     I had it for just a few hours one  
16:42 20 evening to look at it.

21           Q.     Okay.

22           A.     So to say that I have read it does  
23 not imply that I have studied the document --

24           Q.     Uh-huh.

16:42 25           A.     -- or that I have seen enough of

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16:42 1 it to have a really good recollection of that  
2 document. Does that --

3 Q. Yes.

4 A. Okay.

16:42 5 Q. And so I -- my question to you is  
6 simply based on the limited knowledge that you  
7 have of the report and not on any discussions  
8 with counsel, do you agree with what  
9 Mr. Brubaker concluded about your work in '68,  
16:42 10 '69, and '70?

11 A. My recollection is that a large  
12 part I did. There were parts about it that I  
13 did not agree on, as I recall, but I don't  
14 remember that he was unfair or off. That's  
16:43 15 just a recollection from a long time ago.

16 Q. Okay. Several issues that have  
17 kind of arisen from the BRD events is the, I  
18 guess maybe the allegation that -- that you --  
19 your people and RJR came to learn of some --  
16:43 20 some risks that related to tryptophan and how  
21 tryptophan -- before we talked about the --  
22 how, I think one of your criticisms of the  
23 German studies was there were drugs involved  
24 and that there might be adverse relationships  
16:43 25 between the smoking and the drugs administered

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:43 1 by the scientists.

2 Did you come to learn anything  
3 about tryptophan and how it might relate to  
4 smoking during those years?

16:44 5 A. I don't recall a thing about  
6 tryptophan. That's not to say that I didn't  
7 know something at the time. I just don't  
8 recall it right now. Maybe if I saw something  
9 that would prompt my memory, it would bring it  
16:44 10 back. But I just do not recall the  
11 tryptophane story at all.

12 Q. Okay. What about polonium 210?

13 A. I don't know anything about it.  
14 Sorry.

16:44 15 Q. We've talked about really one big  
16 issue today, smoking and health in general.  
17 And I've gone through your report and we've  
18 talked about your basic opinions with regard  
19 to the state of the scientific evidence.

16:45 20 A. Yes, sir.

21 Q. We have also talked just briefly  
22 about the statements that are in your opinions  
23 regarding the BRD issues.

24 A. Yes, sir.

45 25 Q. Are there any other areas that you

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16:45 1 have been asked to give -- offer opinions  
2 about in this case that we haven't addressed  
3 and that aren't addressed in the statement  
4 which is marked as Exhibit 1?

16:45 5 A. Not that I -- not that I recall.

6 Q. Okay. And have you formulated any  
7 other opinions that you intend to offer at  
8 trial if you were called to trial?

9 A. I can't think of any.

16:46 10 Q. Okay.

11 MR. THOMPSON: Let's go off  
12 the record.

13 THE VIDEOGRAPHER: Off the  
14 video record.

15 (Off-the-record  
16 discussion.)

17 THE VIDEOGRAPHER: On the  
18 video record.

19 Q. (By Mr. Thompson) Doctor, will  
16:46 20 you look at what I've previously marked as  
21 Exhibit 5?

22 A. Yes, sir.

23 Q. And very quickly, it appears to be  
24 a collection of, I guess, hard copy of slides,  
16:46 25 a slide series that involved a presentation in

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:46 1 1988 to a tobacco marketing conference.

2 A. Can I look through it just a  
3 second?

4 Q. You can.

16:47 5 A. That is correct.

6 Q. Okay. And just very briefly, can  
7 you tell me what the purpose of this  
8 presentation was?

9 A. I was asked by the then director  
16:47 10 of research, Dr. Robert Demarco, if I would  
11 make a presentation on my literature review to  
12 this marketing group that was at Hilton Head  
13 Island.

14 Q. Was it just RJR marketing people?

16:47 15 A. Yes, it was.

16 Q. And the slides that you produced,  
17 I flipped through them in some detail, and  
18 they look like they kind of recap some of the  
19 issues that you and I have already discussed  
16:48 20 today, which is some of the anomalies and the  
21 epidemiology and also some issues that relate  
22 to nicotine.

23 A. That is correct, yes, sir.

24 Q. Since 1988, have you -- have you  
16:48 25 updated or created a revised version of this

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16:48 1 presentation?

2 A. I have not. I have -- I have  
3 continued to accumulate papers, but I have not  
4 prepared another version of this particular  
16:48 5 presentation, no.

6 Q. And in terms of -- and this is  
7 something I just need to try and get an  
8 understanding of. I was provided with a box  
9 or a pile of medical articles, scientific  
16:48 10 journal articles, and told that those were  
11 the -- the documents on which you would base  
12 your opinions in this case.

13 Did you provide those documents to  
14 the lawyers in this case?

16:49 15 A. I did. And they are -- they are  
16 representative articles. I mean, it does not  
17 exhaust, by any stretch of the imagination, my  
18 holdings on any one of those subjects.

19 Q. Okay.

16:49 20 A. They would be sort of  
21 representative.

22 Q. And I understand what you're  
23 saying. Here's my concern, and I just want to  
24 make sure it's on the record so there's no  
16:49 25 misunderstanding. There's a lot of -- there's

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16:49 1 thousands of articles out there on smoking and  
2 health. And in going through the articles you  
3 provided, there are kind of some highlights.  
4 We've got everything from Dahl in '50, to, you  
16:49 5 know, some outtakes from the surgeon general's  
6 reports.

7 What I need to ask you, sir, is,  
8 if there turns up to be a new article or a  
9 different article and you intend to come to  
16:49 10 trial and put it in a slide show or talk to  
11 the jury about it, I would ask you to please  
12 supplement those -- those articles, because as  
13 we stand here today, I'm going to assume that  
14 these are the articles that you're going to  
16:50 15 talk about unless we see something else.

16 A. I certainly have no objection  
17 personally. I mean, I don't -- I don't know  
18 what the legal problems are.

19 MR. THOMPSON: You know what  
16:50 20 I'm looking for.

21 MS. FORBES: Jeff, I think we  
22 can work it out. I mean, for example, you  
23 made reference to these two abstracts. If  
24 upon receiving them when they are available he  
16:50 25 had developed additional opinions, finalized

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16:50 1 statistics, of course we would provide those  
2 statistics to you.

3 MR. THOMPSON: Absolutely.

4 And I just want to make sure that we're clear  
16:50 5 on that. With that, I think I'm done. I pass  
6 the witness.

7 MS. FORBES: I have no  
8 questions.

9 THE VIDEOGRAPHER: Off the  
16:50 10 video record.

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STATE OF TEXAS

COUNTY OF DALLAS

I, AMY DOMAN, a Certified Shorthand Reporter duly commissioned and qualified in and for the State of Texas, do hereby certify that there came before me on the 15th day of August, 1997, in the offices of Womble, Carlyle, Sandridge, & Rice, located at 200 West Second Street, in the City of Winton-Salem, State of North Carolina the following named person, to-wit: **WILLIAM SAM SIMMONS**, who was duly sworn to testify the truth, the whole truth, and nothing but the truth of knowledge touching and concerning the matters in controversy in this cause; and that he was thereupon examined upon his oath and his examination reduced to typewriting under my supervision; that the deposition is a true record of the testimony given by the witness, and signature of witness is to be before any notary public.

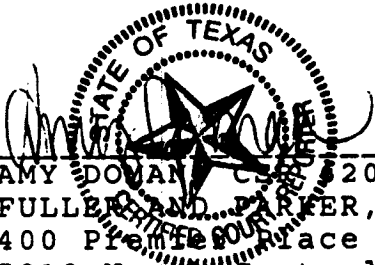
I further certify that I am neither attorney or counsel for, nor related to or employed by any of the parties to the action in which this deposition is taken, and further that I am not a relative or employee of any attorney

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or counsel employed by the parties hereto, or  
financially interested in the action.

GIVEN UNDER MY HAND AND SEAL OF  
OFFICE, on this 28th day of  
August, 1997.

  
AMY DOVAN, Clerk, 203  
FULLER AND PARKER, INC.  
400 Premier Place  
5910 North Central Expressway  
Dallas, Texas 75206

Charge for transcript and exhibits \$ \_\_\_\_\_

To be paid by Plaintiff/Kaiser

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# CORRIGENDUM

Asg No 13396

**[Disregard if signature waived]**

**PAGE**

**LINE**

**CHANGE/REASON**

**Signature of Witness**

**STATE OF TEXAS**

**COUNTY OF**

**SUBSCRIBED AND SWORN TO by the said witness,**

\_\_\_\_\_, on this the \_\_\_\_\_ day of \_\_\_\_\_

**My commission expires:**

**Notary Public in and for the State of**

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Lawyer's Notes

**LIST OF OPINIONS OF DR. WILLIAM SAMUEL SIMMONS  
PURSUANT TO CASE MANAGEMENT ORDER PARAGRAPH 7(B)(1)**

Defendant R.J. Reynolds Tobacco Company (RJRT) submits the following anticipated opinions of Dr. William Samuel Simmons, as required by Paragraph 7(b)(1) of the Case Management Order, as amended.

1. Dr. Simmons is expected to testify that in general, four lines of scientific evidence have been considered in making judgments on the causal relationship between smoking and various diseases. They are: (1) epidemiology; (2) toxicology; (3) chemistry; and (4) mechanism. Dr. Simmons is further expected to testify that, as noted in the 1964 Surgeon General's Report, causation in the area of chronic diseases associated with smoking is a matter of judgment based on an analysis of scientific evidence. ("Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service," 1964.)

2. Dr. Simmons is expected to testify that a statistical association is only one of the lines of evidence that must be considered in making a scientific judgment as to whether a substance causes lung cancer. Although cigarette smoking has been epidemiologically associated with, and therefore is, a risk factor for certain chronic diseases in humans, it has not been scientifically established that smoking causes disease in humans. Association does not establish causation. Further, Dr. Simmons is expected to testify that epidemiologic studies have numerous weaknesses in comparison to controlled laboratory animal experiments.

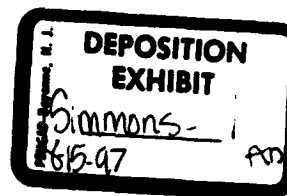
3. Dr. Simmons is expected to testify that positive animal data is an important consideration in making judgments concerning disease causation. In spite of extensive testing with various animal species and strains, scientists have been unable to induce, in laboratory animals appropriately exposed to fresh whole smoke, lung cancer or the other principal diseases associated with smoking in humans. ("The Health Consequences of Smoking: Cancer, A Report of the Surgeon General," 1982, p. 218.)

4. Dr. Simmons is expected to testify that the mechanism or mechanisms of the induction of chronic, noninfectious, long latent period diseases, such as cancer, cardiovascular disease and obstructive pulmonary disease are not known, and that no constituent of smoke, as found in smoke, has been found to cause such diseases.

5. Dr. Simmons is expected to testify that based on the lack of supporting toxicological data and the lack of evidence of a mechanism, it is his opinion that causation has not been conclusively established.

6. Dr. Simmons is expected to testify that the diseases associated with smoking in humans occur in both smokers and nonsmokers. Dr. Simmons is further expected to testify that many other factors have been reported to be statistically associated with the diseases associated with smoking, including, but by no means limited

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to gender, race, age, stress, urban residence, air pollution, lack of exercise, environmental factors, dietary factors, certain occupations or occupational exposures, prior health history and family health history.

7. Dr. Simmons is expected to testify that in 1968, he was hired by Reynolds to work as a chemist in the Biological Research Division (BRD). His research included exposing animals to smoke and analyzing changes within their lungs. Part of his work focused on developing possible methodologies for exposing animals to smoke so as to have a reliable means of studying a potential mechanism for emphysema. Specifically, their plan was to look at the influence of cigarette smoke on pulmonary surfactant to determine whether it was relevant to emphysema.

8. Dr. Simmons expected to testify that there were various problems with the BRD experiments, including the use of only a small number of animals and disease in the animal population. The BRD surfactant work was preliminary. While their findings were not significant and their results were not publishable, he felt free to discuss the work with outside scientists.

9. Dr. Simmons is expected to testify that as a result of the convergence of several events, he was one of numerous scientists discharged by Reynolds in 1970. At the time of his discharge, Reynolds had been ordered by the federal government to divest itself of its starch company, Penick & Ford. At about the same time, Reynolds decided not to pursue the acquisition of a pharmaceutical company. In addition, the group working on the development of a smoking machine for us by CTR had completed their work. As a result of these factors, Reynolds' need for scientists in these fields diminished, and Reynolds reassigned or discharged many of the BRD scientists.

In addition to the opinions listed, Dr. Simmons is expected to comment upon and refer to various studies and other research in support of his testimony. Dr. Simmons may comment upon the opinions expressed by other experts in this case as they relate to issues of causation, and toxicology to the extent that such evidence and opinions are within the scope of his expertise. Defendant specifically reserves the right to elicit from Dr. Simmons opinions in addition to those set forth herein such as opinions that, for example, rebut opinions of plaintiff's expert witnesses or which are statements, beliefs, conclusions or determinations supporting Dr. Simmons' stated opinions.

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SMOKING

AND

TOBACCO

CONTROL

MONOGRAPH

7

The FTC  
Cigarette Test  
Method for  
Determining  
Tar, Nicotine,  
and Carbon  
Monoxide Yields  
of U.S. Cigarettes

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*Report of the NCI Expert Committee*

NATIONAL INSTITUTES OF HEALTH  
National Cancer Institute

DEPOSITION  
EXHIBIT

## Foreword

In response to the emerging scientific evidence that cigarette smoking posed a significant health risk to the user, in the early 1950's the major cigarette manufacturers began widespread promotion of filtered cigarettes to reassure smokers that, regardless of whatever unhealthy constituents were in cigarette smoke, filters were a "scientific" breakthrough.

Advertisements for Viceroy's "health guard filter" stated, "DENTISTS ADVISE—Smoke VICEROYS—The Nicotine and Tars Trapped by The Viceroy Filter CAN NEVER STAIN YOUR TEETH!" and "Leading N.Y. Doctor Tells His Patients What to Smoke—Filtered Cigarette Smoke Is Better For Health. The Nicotine and Tars Trapped . . . Cannot Reach Mouth, Throat Or Lungs." Chesterfield was "Best for you—low in nicotine, highest in quality," while L&M's were "Just What the Doctor Ordered." Lorillard Tobacco Company stressed its science-based Kent micronite filter (the original micronite filter was made of asbestos) and claimed it removed seven times more tar and nicotine than any other cigarette, which "put Kent in a class all by itself where health protection is concerned." Of course, we know today that not only were these claims patently false, but the cigarette companies knew it.

In the early 1950's the Federal Trade Commission (FTC) challenged a variety of health claims made for cigarettes in their advertising, including claims about tar and nicotine. In 1955 FTC published advertising guidelines that, among other things, prohibited claims by cigarette manufacturers that a particular brand of cigarettes was low in tar and nicotine or lower than other brands, when it had not been established by competent scientific proof that the claim was true and the difference was significant. Cigarette manufactures, however, continued to advertise tar numbers. In the absence of a standardized test methodology, this resulted in what is referred to as a "tar derby"—a multitude of inconsistent, noncomparable claims that did not give consumers a meaningful opportunity to assess the relative tar delivery of competing brands. The tar derby ended in 1960 when discussions with FTC culminated in an industry agreement to refrain from tar and nicotine advertising.

In 1966, however, the U.S. Public Health Service (PHS) prepared a technical report on "tar" and nicotine that concluded, "The preponderance of scientific evidence strongly suggests that the lower the 'tar' and nicotine content of cigarette smoke, the less harmful would be the effect." In reaching this conclusion, the report noted the clear relationship between dose of cigarette smoke received by the smoker and disease risk. Regardless of how dose was calculated—by number of cigarettes smoked per day, age of initiation, total number of years one smoked, or depth of inhalation, mortality rates among smokers increased. When smokers quit smoking, their risk was reduced in proportion to the length of time off cigarettes.

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Subsequent to the PHS statement, FTC reversed its decision banning tar and nicotine claims in advertising and established a standardized testing protocol for assessing tar and nicotine yields. Today that protocol is widely known as the FTC test method. In 1980 the protocol was broadened to include measurement of the carbon monoxide yields of cigarettes as well.

The initial protocol adopted by FTC was largely based on the work of U.S. Department of Agriculture chemist C.L. Ogg, as published in the *Journal of the Association of Official Agricultural Chemists* in 1964. It appears, however, that this protocol was based on one person's observations about how people smoked.

Much the same protocol had been proposed by American Tobacco Company researchers in 1936. Writing in the July issue of *Industrial and Engineering Chemistry*, J.A. Bradford and colleagues noted, "The present writer's arbitrarily selected rate is a 35-cc puff of 2-second duration taken once a minute."

However, cigarettes consumed at that time were vastly different from those manufactured and marketed later. In fact, tar and nicotine levels began to decline during the 1950's, concurrent with the mass marketing of filter cigarettes. Market share of filter cigarettes increased from almost zero in 1950 (0.6 percent of the market) to 50 percent by decade's end. Total cigarette sales, which had begun to decline after the first public statements about the hazards of smoking in the early 1950's, rebounded to new highs.

Although filter efficiency may have contributed to some of the reduction in tar/nicotine yields in the 1950's, the decline resulted mostly from less tobacco being used to make filtered as opposed to unfiltered cigarettes. However, during the 1960's and 1970's major cigarette design changes resulted in significantly lower machine-measured cigarette yields. The changes included increased use of ventilated tobacco rods and filters, use of more porous cigarette papers, and increased use of expanded and reconstituted tobacco. Concurrent with these modifications in cigarette design, cigarette manufacturers increasingly made use of additives in manufacturing. Today about 600 different compounds are routinely added to domestic cigarette brands, yet no routine testing is performed to determine whether these compounds pose any additional health risk to the smoker when they are burned in a cigarette.

U.S. market share of cigarettes yielding 15 mg tar or less went from 3.6 percent in 1970 to 44.8 percent by 1980. The sales-weighted average tar and nicotine yields of all U.S. cigarettes are now approximately 12 mg tar and 0.9 mg nicotine. By comparison, sales-weighted yields in the early 1950's were 35 mg tar and 2.5 mg nicotine.

As consumption of low-yield cigarettes began to proliferate, the public health community became concerned that these products were not what they seemed. Increasingly, scientific studies documented that smokers who switched to these low-yield products smoked them differently, thus negating

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the reason many of them changed in the first place—to lower their health risk.

The U.S. Congress also voiced its concern in 1978 when it enacted the Health Services and Centers Act. Section 403 of that legislation directed the U.S. Department of Health and Human Services (DHHS) to conduct a "study or studies of (1) the relative health risks associated with smoking cigarettes of varying levels of tar, nicotine, and carbon monoxide; and (2) the health risks associated with smoking cigarettes containing any substances commonly added to commercially manufactured cigarettes." The Secretary of the Department of Health and Human Services addressed this issue as part of the 1981 Surgeon General's report, *The Health Consequences of Smoking: The Changing Cigarette*. The overall conclusion of that report was clear: "There is no safe cigarette and no safe level of consumption." Although the report did note that smoking cigarettes with lower yields of tar and nicotine reduces the risk of lung cancer to some extent, the benefits are minimal in comparison with giving up cigarettes entirely. Evidence relating to heart disease, other cancers, or chronic obstructive lung disease was not sufficient to permit conclusions to be drawn. As to the accuracy of the FTC test method, the report stated: "The 'tar' and nicotine yields obtained by present testing methods do not correspond to the dosages that the individual smokers receive: In some cases they may seriously underestimate these dosages."

Growing numbers of questions were raised about the accuracy of the FTC test protocol to measure tar, nicotine, and carbon monoxide levels from low-yield cigarettes—questions raised not just by the public health community but also within the tobacco industry. Competitors complained to FTC that Brown and Williamson's (B&W) Barclay brand cigarette did not test accurately with the FTC test method. They argued that the brand was designed with unique air ventilation channels that caused it to test low on the FTC method. The ventilation channels, which remained open when Barclays were smoked on the FTC machine, were rendered inoperable when a human being smoked the cigarettes. In April 1983 FTC announced that its testing method understated values for constituents in Barclay cigarettes, and as a result, until new testing methods were developed, FTC would no longer report an official rating for Barclay cigarettes. Later, FTC took similar steps with respect to other B&W cigarette varieties that used a filter design similar to Barclay's.

Eventually FTC closed its cigarette testing laboratory, in part because of insufficient expertise within the agency to carry out an increasingly complex and costly testing program. Since 1987, constituent levels for domestic cigarette brands have been determined for the manufacturers by the Tobacco Institute Testing Laboratory with oversight by FTC. The Tobacco Institute serves as a trade organization as well as the information and lobbying arm of the tobacco industry.

In June 1994 the Chairman of the House Subcommittee on Health and the Environment wrote the Director of the National Cancer Institute (NCI), asking him to convene a meeting of experts to "... review and make

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recommendations on the accuracy and appropriateness of the Federal Trade Commission's method for determining the relative 'tar' and nicotine content of cigarettes." A similar request was received from the FTC Chairman asking that NCI convene a consensus conference on the topic and outlining several areas it wished to be considered.

On December 5 and 6, 1994, a meeting of the NCI ad hoc expert committee was convened under the aegis of the President's Cancer Panel to examine this issue. The committee consisted of 11 individuals from diverse scientific backgrounds and experience. The committee had the benefit of excellent presentations from 14 experts whose professional careers were not only involved in research on smoking, but who have been active contributors to this field of scientific inquiry. Two of the individual participants were cigarette industry scientists, who participated in all discussions.

From the outset of the committee's deliberations, it was clear that the intent of the meeting was not to redesign the FTC testing protocol but, rather, to examine the protocol and make suggestions for improvement, if warranted. To provide a framework for discussion, the committee was asked to consider three basic questions:

- 1. Does the evidence presented clearly demonstrate that changes are needed in the current FTC protocol for measuring tar, nicotine, and carbon monoxide? If yes, what changes are required?**
  - 2. Should constituents other than tar, nicotine, and carbon monoxide be added to the protocol?**
  - 3. Does the FTC protocol provide information useful to smokers in making decisions about their health?**
- I. The committee reached the following conclusions with respect to the first question.
- A. The smoking of cigarettes with lower *machine-measured* yields has a small effect in reducing the risk of cancer caused by smoking, no effect on the risk of cardiovascular diseases, and an uncertain effect on the risk of pulmonary disease. A reduction in *machine-measured* tar yield from 15 mg tar to 1 mg tar does not reduce relative risk from 15 to 1.
  - B. The FTC test protocol was based on cursory observations of human smoking behavior. Actual human smoking behavior is characterized by wide variations in smoking patterns, which result in wide variations in tar and nicotine exposure. Smokers who switch to lower tar and nicotine cigarettes frequently change their smoking behavior, which may negate potential health benefits.
  - C. Accordingly, the committee recommends the following changes to the FTC protocol:

1. This system should also measure and publish information on the range of tar, nicotine, and carbon monoxide yields that most smokers should expect from each cigarette sold in the United States.
  2. This information should be clearly communicated to smokers.
  3. A simple graphic representation should be provided with each pack of cigarettes sold in the United States and in all advertisements. The representation should not imply a one-to-one relationship between measurements and disease risk.
  4. The system must be accompanied by public education to make smokers aware that individual exposure depends on how the cigarette is smoked and that the benefits of switching to lower yield cigarettes are small compared with quitting.
- D. There should be Federal oversight of cigarette testing, but such testing should continue to be performed by the tobacco industry and at industry expense.
- E. The questions involved in the purpose, methodology, and utility of the FTC protocol are complex medical and scientific issues that require ongoing involvement of Federal health agencies, including the National Institutes of Health, the Food and Drug Administration, and the Centers for Disease Control and Prevention.
- F. The system should be reexamined at least every 5 years to evaluate whether the protocol is maintaining its utility to the smoker.
- G. When a cigarette manufacturer makes significant changes in cigarette design that affect yields, it should notify the appropriate Federal agency.
- II. With regard to the second question, the committee recommends that to avoid confusing smokers, no smoke constituents other than tar, nicotine, and carbon monoxide be measured and published at the present time. Smokers should be informed of the presence of other hazardous smoke constituents with each package and with all advertisements. These constituents should be classified by toxic effects.
- III. In considering the third question, the committee reached the following conclusions:
- A. Information from the testing system is useless to smokers unless they have ready access to it. The information from the testing system should be made available to all smokers, including those who smoke generic brands and other brands not widely advertised.
  - B. Brand names and brand classifications such as "light" and "ultralight" represent health claims and should be regulated and accompanied, in fair balance, with an appropriate disclaimer.

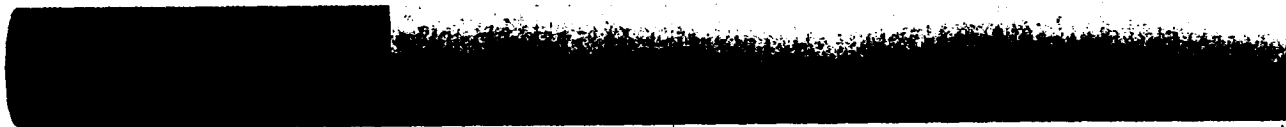
- C. The available data suggest that smokers misunderstand the FTC test data. This underscores the need for an extensive public education effort.

As Chairman of the President's Cancer Panel under whose aegis this meeting was convened, I would like to express here my admiration and deep appreciation to the members of the NCI ad hoc committee and its expert consultants for a job well done. In transmitting this report to both the U.S. Congress and the Federal Trade Commission, it is my sincere hope that the recommendations contained herein will receive the serious and thoughtful consideration they deserve.

Harold P. Freeman, M.D.  
Chairman, President's Cancer Panel

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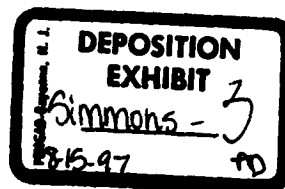
**Yours have additives.\***



**94% tobacco  
6% additives**

\*Laboratory analyses of the top ten U.S. non-menthol brand styles show all of their tobaccos contain a minimum of 6% additives on a dry weight basis.

**SURGEON GENERAL'S WARNING: Cigarette  
Smoke Contains Carbon Monoxide.**



16 mg. "tar", 1.1 mg. nicotine  
av. per cigarette by FTC method.

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New Winstons don't.



**100% tobacco  
True taste**



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**PHILIP MORRIS U.S.A.**

**NEWS RELEASE**

Corporate Affairs  
120 Park Avenue  
New York, New York 10017  
(212) 880-5000  
FAX (212) 907-5361

FOR IMMEDIATE RELEASE  
April 13, 1994

CONTACT:  
Victor Han  
(202) 637-1566

**SIX MAJOR AMERICAN CIGARETTE COMPANIES RELEASE  
COMBINED INGREDIENTS LIST**

New York -- Philip Morris U.S.A., along with the five other major American cigarette manufacturers, today approved the release of the industry's combined list of ingredients added to tobacco in cigarettes manufactured and sold in the United States.

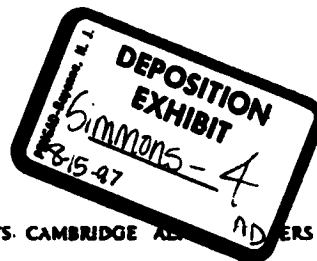
"The industry's decision to make this proprietary information publicly available to our consumers is in response to misleading allegations recently made about the nature of the ingredients used in our products," said Steven Parrish, senior vice president and general counsel for Philip Morris U.S.A.

He said the six major American cigarette manufacturers decided to voluntarily disclose the combined ingredients list to "demonstrate to our consumers that cigarette ingredients are similar to ingredients used in a wide variety of consumer products and are not harmful to smokers."

Parrish said that Philip Morris and the other five companies had been submitting a list of ingredients added to tobacco used in cigarettes manufactured and sold in the United States to the Secretary of the Department of Health and Human Services (HHS) as required by the Federal Cigarette Labeling and Advertising Act each year since 1986. The Act recognizes that cigarette ingredients are trade secrets and requires that HHS maintain strict confidentiality.

\* "Unfortunately," Parrish said, "the confidentiality that Congress mandated for cigarette ingredients information has been mischaracterized as an attempt by cigarette manufacturers to be 'secretive' and keep information from the American public."

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MARLBORO BENSON & HEDGES MERIT VIRGINIA SLIMS PARLIAMENT LIGHTS CAMBRIDGE ALLIANCE SARATOGA

In addition to releasing the ingredients list, the companies made available a copy of a March 1994 report, "A Safety Assessment of Ingredients Added to Tobacco in the Manufacture of Cigarettes." Parrish said the independent assessment, performed by six eminent scientists, had been conducted on the entire list of cigarette ingredients provided to HHS by the six major American cigarette manufacturers. The authors concluded that "the ingredients added to tobacco in the manufacture of cigarettes by United States manufacturers are not hazardous under the conditions of use."

According to Parrish, all of the ingredients added to tobacco used in cigarettes manufactured and sold in the United States by Philip Morris are common foods or food additives, and are included on the Food and Drug Administration's lists of approved food additives on substances "generally recognized as safe" (GRAS), are on the Flavor Extract Manufacturers Association's GRAS list, or have been approved by federal agencies such as the Bureau of Tobacco, Alcohol and Firearms or the Environmental Protection Agency.

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UPI 04/13 1632 Cigarette industry releases list of 600 ingredients

WASHINGTON, April 13 (UPI) -- The six major U.S. tobacco companies released Wednesday the list of 600 ingredients they add in manufacturing cigarettes in a move to combat pressure for more regulation of their industry.

R.J. Reynolds Tobacco Co. revealed the ingredients on behalf of the major U.S. cigarette manufacturers, including the American Tobacco Co., Brown and Williamson, Liggett Group, Inc., Lorillard, Inc., and Philip Morris Inc.

Reynolds spokesman David Fishel said, "More than 98 percent of the ingredients are approved as food additives by the U.S. Food and Drug Administration, and have been given the status 'Generally Recognized as Safe in Foods' by the FDA or other expert committees."

Critics, however, said some of the ingredients were toxic, and Rep. Ron Wyden, D-Ore., said about 100 ingredients were missing from the list that had been formerly disclosed to federal authorities.

Philip Morris spokesman Tony Andrade said, "The 600 ingredients is the complete list given to the federal government under the Federal Cigarette Labeling Act. There might be a few other ingredients added to the paper because the law does not refer to ingredients added to cigarette paper."

Congressional hearings are scheduled for Thursday to further investigate the cigarette industry.

Andrade said the ingredients list was not released in anticipation of congressional hearings, but he expected discussion of the ingredients to be on the agenda.

Among the primary ingredients, in addition to tobacco, are water, sugar, glycerin, licorice, cocoa and additional flavorings. The list of 600 ingredients, agents and chemicals that are added to the primary items during manufacture run the alphabet from acetanisole to yeast.

Some of the additives include alfalfa, ammonia, ascorbic acid, basil and bay leaf oil, caffeine, carbon dioxide, beta-carotene, ethyl alcohol, ethyl propionate, honey, smoke flavor, snakeroot oil, vanilla, wild cherry bark, wine and xanthan gum.

Cigarettes are 90 percent tobacco, Reynolds said. Other primary ingredients of 99 percent of U.S. non-menthol cigarettes also contain water, sugars, glycerin, propylene glycol, licorice, cocoa and additional flavors. Additional ingredients, the company said, for flavoring make up about 0.02 percent of cigarettes.

The Action on Smoking and Health, however, said some of the chemicals are so toxic that they could not be dumped in a landfill under federal environmental laws.

John Banzhaf, director of the anti-smoking organization, said, "It is unconscionable that the industry would be able to add chemicals too dangerous to be used in foods or even added to landfills with any governmental testing."

Banzhaf also said the ingredient list includes at least 13 chemicals "which are banned by the FDA because they are too dangerous to be allowed in foods."

The anti-smoking organization cited ethyl-2-fluoroate, which causes liver damage in testing on animals; freon-11, a chlorofluorocarbon; and methoprene, a pesticide used to kill insects on stored tobacco.

Pressure has been mounting recently for the tobacco industry to be more forthcoming in information on the manufacture of cigarettes. Testimony

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during March hearings chaired by Rep. Henry Waxman, D-Calif., for the Energy and Commerce Committee's subcommittee on health and the environment, suggested tobacco companies hid information about cigarette ingredients and knew that nicotine is addictive. Waxman has suggested further regulation of cigarette manufacturing.

Waxman said he also is considering legislation that would authorize the Food and Drug Administration, which currently has virtually no oversight power over the tobacco industry, to regulate cigarettes as a drug delivery system.

Waxman said he would hold a hearing of his subcommittee Thursday to investigate such matters and had sent "formal letters of invitation" to the chief executive officers and scientific research directors of Philip Morris and several other tobacco companies.

Philip Morris earlier said they were releasing the list of ingredients to show they are not harmful to smokers.

"The industry's decision to make this proprietary information publicly available to our consumers is in response to misleading allegations recently made about the nature of the ingredients used in our products," said Steven Parrish, general counsel for Philip Morris U.S.A.

Parrish said that Philip Morris and the other five companies had been submitting a list of ingredients added to tobacco used in cigarettes manufactured and sold in the United States to the Secretary of the Department of Health and Human Services (HHS) as required by the Federal Cigarette Labeling and Advertising Act each year since 1986.

The Act recognizes that cigarette ingredients are trade secrets and requires that HHS maintain strict confidentiality.

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"Unfortunately," Parrish said, "the confidentiality that Congress mandated for cigarette ingredients information has been mischaracterized as an attempt by cigarette manufacturers to be 'secretive' and keep information from the American public."

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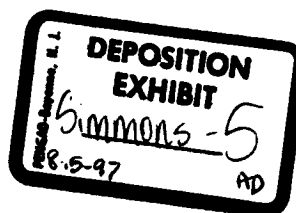
PRESENTATION BY  
WILLIAM S SIMMONS  
TO

1988 TOBACCO MARKETING CONFERENCE

MAY 24, 1988

HILTON HEAD

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R & D (S & H  
Presentation to  
Marketing Conf -  
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Slide: JW001

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 720 Mon May 16 04:39:56 1988

# Smoking and Health Division

reports directly to  
Dr. G. Robert DiMarco

01000002

# Anomalies in Lung Cancer Epidemiology

- 1 Diagnostic Bias Against Nonsmokers
- 2 Declining Risk in Males
- 3 High Rates in Nonsmoking  
Chinese Women

01000003

## Diagnostic Bias Against Nonsmokers

"The correct diagnosis had not been made during life in 26% of 153 patients with lung cancer found in necropsies performed between 1971 and 1982. . . ."

(Archives Internal Medicine, 1986)

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. Slide: JW003A2 Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 364 Mon May 16 04:51:56 1988

## Diagnostic Bias Against Nonsmokers

"Furthermore, if a lesion was present, chest films were more likely to be radiologically interpreted as a cancer in smokers."

(Archives Internal Medicine, 1980)

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Slide: JW004

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 366 Mon May 16 04:54:07 1988

## Declining Risk in Males

Age-adjusted incidence and mortality rates due to lung cancer have been decreasing for both white and black males in certain age groups since 1973.

(JAMA, Vol. 256, 1986)

01000006

Slide: JW005A    Opid: Simmons Mktg    Job #: 51488    DDS:6000    size: 430    Mon May 16 04:58:32 1988

## High Rates in Nonsmoking Chinese Women Hong Kong

- Lung cancer is the major cause of death in males and females. Half of the female lung cancer patients are nonsmokers.

(Lam et al., 1987)

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Slide: JW006A Opid: Simmons Mktg Job #: 51488 DDS:6000 size: 440 Mon May 16 05:03.42 1988

## High Rates in Nonsmoking Chinese Women Shanghai, P.R.C.

- One of highest female lung cancer rates in the world (20 per 100,000)
- Only 25% of new cases occur in cigarette smokers.

(Gao et al., 1987)

01000008

Slide: JW007A    Opid: Simmons Mktg    Job #: 51488    DDS:6000    size: 428    Mon May 16 05:08:37 1988

High Rates in  
Nonsmoking Chinese Women  
Auen Wei County, P.R.C.  
(pop. 1,000,000)

- Lung cancer in women is 323%  
higher than in U.S.
- Only 0.1% of the women smoke.

(Murnford et al., 1987)

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Slide: JW008

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 292 Mon May 16 05:11:03 1988

## **Anomalies in Heart Disease Epidemiology**

**Smoking is not associated with heart  
disease in countries where a low-fat  
diet is consumed.**

**(McGill, 1987)**

0100000A

Slide: JW009

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 406 Mon May 16 05:12:31 1988

## Anomalies in Heart Disease Epidemiology

In Western countries, smokers have been reported to have only slight to moderate increases in coronary artery atherosclerosis (20-30%) compared to nonsmokers.

(McGill, 1987)

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Slide: JW0010 Opid: Simmonns Mktg

Job #: 51488 DDS:6000 size: 300 Mon May 16 05:14:47 1988

# Anomalies in Heart Disease Epidemiology

Smokers have been reported to have  
slightly lower blood pressure than  
nonsmokers.

(Surgeon General's Report, 1983)

0100000C

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Slide: JW011

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 268 Mon May 16 05:16:52 1988

## Exercise and Heart Disease

Recent studies have suggested  
an increasingly important role  
for exercise in the prevention  
of coronary heart disease.

0100000D

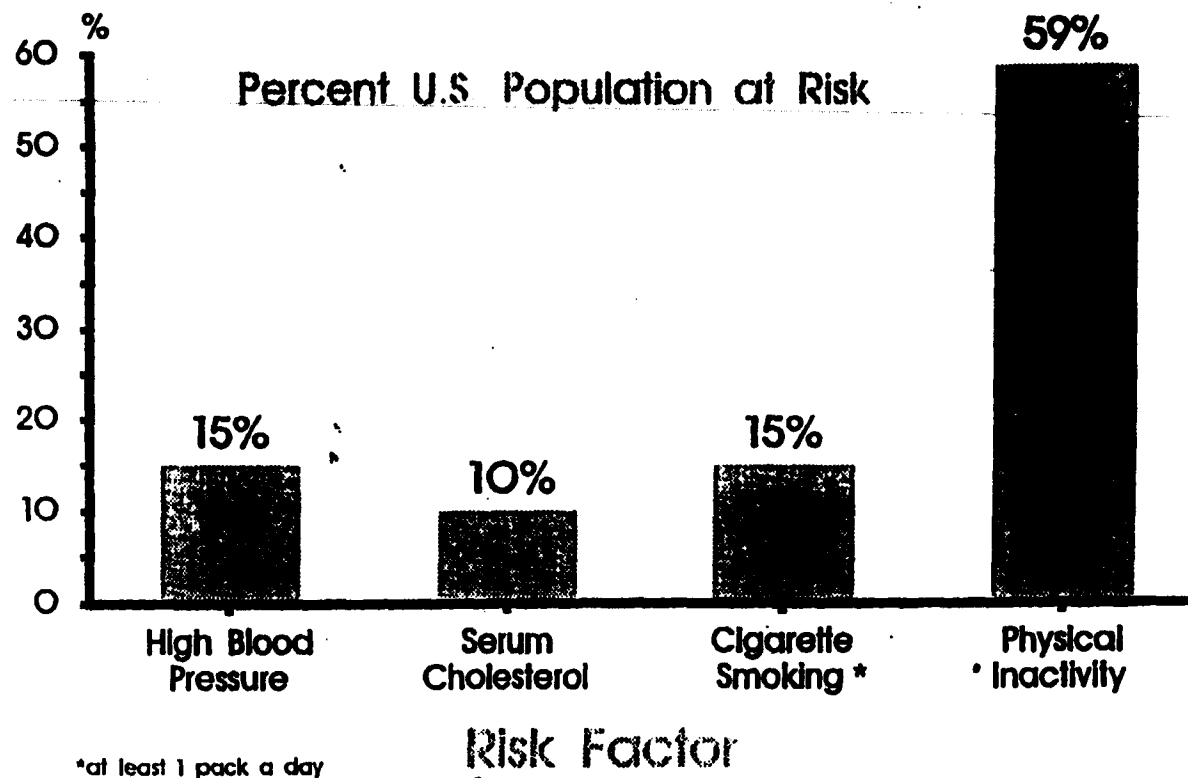
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Slide: JW012-U Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 1336 Mon May 16 05:18:38 1988

## Recognized Risk Factors Related to Coronary Heart Disease



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Slide: JW013A Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 422 Mon May 16 05:28:15 1988

## **Carbon Monoxide and Cardiovascular Disease**

**"There is no longer evidence for  
considering carbon monoxide to be a  
component of major importance for the  
enhanced atherosclerosis in tobacco  
smokers. . . ."**

**(Hugod and Astrup, 1981)**

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Slide: JW014

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 1854 Mon May 16 05:31:00 1988

## Positive Aspects of Nicotine Use

- 1 Enhanced Mental Performance
- 2 Decreased Anxiety
- 3 Reduced Aggression
- 4 Increased Tolerance to Pain
- 5 Body Weight Control

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Slide: JW015

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 614 Mon May 16 05:33:47 1988

# Enhanced Mental Performance

## ■ Enhanced Processing of Sensory Information

(Warwick and Eysenck, 1968)

## ■ Increased Vigilance

(Wesnes and Warburton, 1978)

## ■ Enhanced Rapid Information Processing

(Wesnes and Warburton, 1983)

## ■ Improvement in State-dependent Learning

(Warburton, Wesnes, Shergold, 1982)

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Slide: JW016

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 316 Mon May 16 05:39:28 1988

## Decreased Anxiety

Anxiety induced by having subjects attempt to solve a difficult puzzle was reduced by smoking a usual-nicotine delivery cigarette.

(Pomerleau, 1986)

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Slide: JW017

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 202 Mon May 16 05:41:24 1988

## Reduced Aggression

Nicotine has been reported  
to reduce aggression in squirrel  
monkeys, rats, and cats.

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Slide: JW017ALT Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 432 Mon May 16 05:42:56 1988

# Nicotine reported to reduce aggression in:



## Squirrel Monkeys

(Hutchinson and Emley, 1973)



## Rats

(Driscoll and Boettig, 1981)



## Cats

(Bernston, Beattie, Walker, 1976)

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Slide: JW018

Opid: Simmons Mkg

Job #: 51488 DDS:6000 size: 272 Mon May 16 05:44:45 1988

## Increased Tolerance to Pain

Human subjects have demonstrated increased endurance to pain after smoking usual-nicotine cigarettes.

(Pomerleau, 1986)

01000015

Slide: JW019    Opid: Simmons Mktg    Job #: 51488    DDS:6000    size: 384    Mon May 16 05:46:41 1988

# Body Weight Control

■ As a group, smokers weigh less than nonsmokers.

(Grunberg, 1983)

■ Nicotine is probably responsible for this effect.

(Hotstetter et al., 1986).

01000016

51710 1383

50677 3884

Slide: JW020

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 512 Mon May 16 05:48:34 1988

## Parkinson's Disease

- A brain disease characterized by muscular rigidity, tremor, and abnormal slowness of movement.
- Characterized by depletion of the chemical dopamine in the brain.
- Affects approximately 500,000 people in the United States.

01000017

Slide: JW021

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 242 Mon May 16 05:51:46 1988

## PD and Smoking

Smokers are at approximately half  
the risk for developing PD as  
nonsmokers.

(Neurology, Vol. 36, 1986)

01000018

51710 1385

50677 3886

Slide: JW022A1 Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 262 Mon May 16 05:55:23 1988

**PD and Smoking**  
**Possible Protective Mechanisms**  
Nicotine may prevent PD by increasing  
brain dopamine levels. . . .

(Neurology, Vol. 36, 1986)

01000019

Slide: JW022A2 Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 436 Mon May 16 05:57:10 1988

## PD and Smoking

### Possible Protective Mechanism:

Compounds found in cigarette smoke, e.g., quinoline derivatives, may block the action of neurotoxins which cause the neural damage characteristic of PD.

(D'Amato et al., 1987)  
(Ohta et al., 1987)

0100001A

Slide: JW023

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 418 Mon May 16 05:59:36 1988

# Alzheimer's Disease

- A progressive brain disorder of unknown cause
- Characterized by loss of recent memory, difficulty in learning, changes in personality, and difficulty with movement and speech.

0100001B

Slide: JW024

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 412 Mon May 16 06:02:11 1988

## Public Health Impact of AD

- Estimated 2 million Americans currently affected.
- Killed 120,000 Americans last year. (100,000 deaths attributed to lung cancer)

(So. Ill. School of Med., 1987)

0100001C

Slide: JW025

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 466 Mon May 16 06:04:18 1988

## Public Health Impact of AD

- In 1900, 3 million Americans were at risk for AD. By 2000, 30 million people will be at risk.
- 5% of individuals over 65 develop moderate Alzheimer's disease.

(So. Illinois School of Med., 1987)

0100001D

Slide: JW026

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 336 Mon May 16 06:06:29 1988

## Nicotine and Alzheimer's Disease

At least 3 different research groups are currently experimenting with nicotine as a treatment for the attention deficit and memory loss associated with AD.

0100001E

51710 1391

50677 3892

Slide: JW027

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 232 Mon May 16 06:08:47 1988

AD Research Funded by RJR

**Jeffrey Gray**  
(London)

Administering nicotine to AD  
patients for attention deficit.

0100001F

Slide: JW028

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 338 Mon May 16 06:10:26 1988

AD Research Funded by RJR

**Dr. Ezio Giacobini**  
(So. Ill. Univ.)

Studying effect of nicotine on  
receptors. Receptors are  
deficient in AD patients and  
increased in smokers.

01000020

51710 1393

50677 3894

Slide: JW029    Opid: Simmons Mktg    Job #: 51488    DDS:6000    size: 184    Mon May 16 06:12:35 1988

AD Research Funded by RJR  
**Dr. Robert Pfeffer**  
(U. Cal. Irvine)  
Epidemiological Study

01000021

Slide: JW030

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 448 Mon May 16 06:13:54 1988

## Health Effects of Nicotine Use

Direct evidence concerning the carcinogenicity of nicotine is lacking, but the lack of an association between smokeless tobacco use and cancers outside the oral cavity suggests that nicotine is not carcinogenic.

(SGR 1986)

01000022

51710 1395

50677 3896

Slide: JW031

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 314 Mon May 16 06:16:25 1988

## Health Effects of Nicotine Use

- Nicotine is not atherogenic.  
(Schlevelbein, 1979)
- Nicotine does not cause bronchitis.  
(Rogers et al., 1986)

01000023

Slide: JW032

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 1302 Mon May 16 08:33:23 1988

## Smoking & Stroke

Study	Reference	Cohort Studied	Study Length	Result
Italian Study	Acta Cardiologica, 1987	1661 Men	25 years	-Mean blood pressure predictive. -No significant smoking effect.
Swedish Study	N.E.J. Med., 1987	792 Men	18.5 years	-Maternal history was a strong predictor (RR=3). -Smoking not a significant risk factor.
Finnish Study	Stroke, 1982	3750 Men 4074 Women	7 years	-Smoking was a statistically significant risk factor in men only.

01000024

51710 1397

50677 3898

Slide: JW033

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 1416 Mon May 16 08:37:23 1988

## Smoking & Stroke (cont'd.)

Study	Reference	Cohort Studied	Study Length	Result
Framingham	JAMA, 1988	4255 Men and Women	26 years	-Relative risk for male smokers = 1.42.  -Relative risk for female smokers = 1.61
Honolulu Heart Program	N.E.J. Med., 1986	8006 Japanese Men	12 years	-Relative risk for smokers was 2-3.
Lipid Research Clinics	Am.J.Epl., 1986	1491 Men 1924 Women (whites)	9 years	-Twofold excess stroke risk in women with family history.

01000025

51710 1398

50677 3899

Slide: JW034 Opid: Simmons Mktg Job #: 51488 DDS:6000 size: 374 Mon May 16 08:31:22 1988

## Smoking and Stroke Italian Study

1661 Men - 25 Years

### Results

- Mean blood pressure significant
- No significant smoking effect.

(Acta Cardiologica, 1987)

01000026

Slide: JW035    Opid: Simmons Mktg    Job #: 51488    DDS:6000    size: 460    Mon May 16 08:41:40 1988

Smoking and Stroke

# Framingham Study

4225 Men - 26 years  
and Women

## Results

■ Relative risk for male smokers  
= 1.42.

■ Relative risk for female smokers  
= 1.61.

(JAMA, 1988)

01000027

# Smoking and Stroke Swedish Study

792 Men - 18.5 Years

## Results

- Maternal history was a strong predictor ( $RR=3$ ).
- Smoking not a significant risk factor.

(N.E.J. Med., 1987)

01000028

Slide: JW037 Opid: Simmons Mktg Job #: 51488 DDS:6000 size: 354 Mon May 16 08:45:47 1988

# Smoking and Stroke Honolulu Heart Program

8006 Men - 12 Years  
Japanese

## Results

■ Relative risk for smokers  
was 2.3.

(N.E.J. Med., 1986)

01000029

.Slide: JW038

Opid: Simmons Mktg

. Job #: 51488 DDS:6000 size: 394 Mon May 16 08:47:19 1988

## Smoking and Stroke Finnish Study

3750 Men - 7 Years  
4074 Women

## Results

■ Smoking was a statistically significant risk factor in men only.

(Stroke, 1982)

0100002A

Slide: JW039

Opid: Simmons Mktg

Job #: 51488 DDS:6000 size: 406 Mon May 16 08:49:08 1988

Smoking and Stroke

## Lipid Research Clinics

1491 Men - 9 Years

1924 Women

(Whites)

## Results

■ Twofold excess stroke risk  
in women with family history.

(Am.J.Epi., 1986)

0100002B

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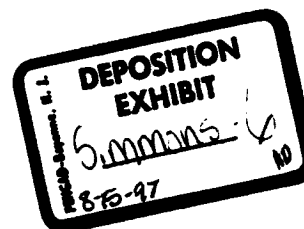
on

**The R. J. Reynold's Tobacco Company's  
Biology Research Division****A Program Review****1965 - 1970****Prepared for****R. F. McDermott, Esq.  
J. C. McElvain, Esq.  
C. Bordenave, Esq.**

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201-543-6108****December 15th, 1985****CONFIDENTIAL**

51710 1404

50792 8501

10359

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